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TORSION BALLISTOCARDIOGRAPHY IN CARDIOVASCULAR SURGICAL PATIENTS

C. FREDERICK KITTLE, M.D.* (BY INVITATION), KURT R. REISSMANN, M.D. (BY INVITATION), E. GREY DIMOND, M.D., (BY INVITATION), AND PAUL W. SCHAFER, M.D.

KANSAS CITY, KAN.

CONCERTED interest in cardiovascular diseases, undoubtedly aroused by tremendous strides in the diagnosis and surgical treatment of these conditions, has occasioned the exploitation of varied biophysical methods for better understanding of the basic factors in cardiovascular physiology. Ballistocardiography, although recognized and described in 1877 by Gordon,[1] remained a relatively unknown technique until recent years. After interest was stimulated in this subject by the fundamental work of Starr in 1939,[2] numerous investigators have devoted themselves to its study.[3-8] Ballistocardiography is a method of recording body movement caused by the acceleration and deceleration of blood as it is ejected and moved about in blood vessels, and to a certain extent by the motion of the heart. Our interest in this field has involved study of the ballistocardiogram in patients subjected to cardiovascular surgery, as recorded by a particular type of apparatus termed the torsion ballistocardiograph. In these patients it has been possible to correlate known anatomic defects and surgical alterations with ballistocardiograms before and after operation. Since this review is based on serial records of the same patient, many problems inherent in the comparison of ballistocardiograms in different patients are eliminated.

METHOD

The most important factors causing the ballistic waves are the acceleration produced by cardiac ejection of blood and its subsequent deceleration in the great vessels and arterioles. Although right and left ventricular con-

From the Departments of Surgery and Medicine, The University of Kansas Medical Center.

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*Markle Scholar in medical sciences.

tractions are the primary forces in acceleration of the blood, numerous variables influence deceleration: the dimensions of the aorta, the elasticity of the aorta, the site of deceleration, and arteriolar drainage.[9-13] The final ballistocardiogram is the summation of all these accelerating and decelerating forces. All of these forces are vectors, that is, they are characterized by both direction and amplitude, and their direction depends upon the topographic anatomy of the heart and great vessels.

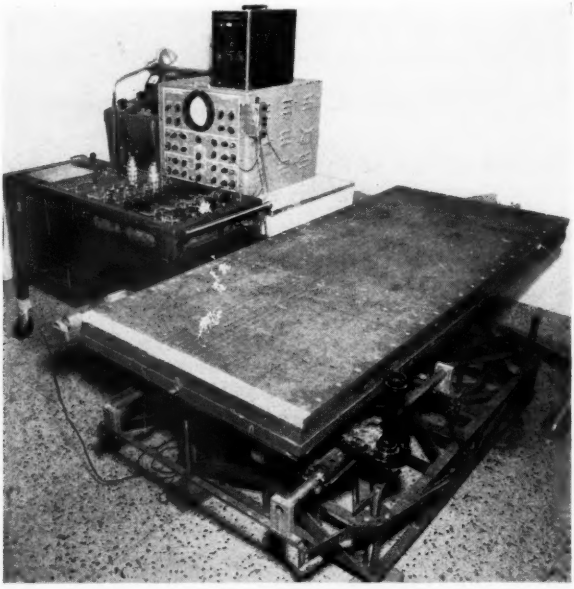

Fig. 1.—The torsion ballistocardiograph. The table is in the foreground while the recording and amplifying devices are seen in the background.

Because these forces are vectors, the torsion ballistocardiograph was devised in an attempt to delineate their direction and their origin. The torsion ballistocardiograph consists of a table top supported only by an upright steel bar in its center (Fig. 1). This steel bar because of its torsion elasticity permits circular movements of the table. Forces along axes through the center of rotation cause no displacement of the table. The magnitude of any circular displacement of this table is proportional to the product of the force times its distance from the center of motion (lever arm). When the force acts farther away from the center, a proportionally greater displacement is present than when it acts closer. By positioning a patient in relation to the center of motion, reaction forces in various parts of the cardiovascular system can be accentuated or attenuated. By noting the position of the patient where a given ballistic complex is greatest, where it is absent, and where it is reversed, it is possible to predict approximately its anatomic site of origin and its direction of flow. Knowledge of these factors is more important in the study of congenital cardiovascular defects than in other types of cardiovascular disease.

The technical details of construction and the physical characteristics of this torsion ballistocardiograph have been presented in previous reports.[14-15] The rotational displacement of the table is converted into proportional voltage utilizing condenser plates in a carrier frequency circuit, one plate being stationary, the other being mounted to the table top. The rectified output is amplified by a DC amplifier and recorded by a Sanborn Polyviso machine. A frequency response for the table of $\frac{1}{2\pi f}$ was selected for reasons discussed elsewhere.[14-15] This frequency response in the range from 1 to 20 cycles per second was secured by using a table with a natural frequency of 4 cycles per second and supercritically damping it with a piston moving in an oil-filled cylinder. Changes in the momentum of inertia of the system with people of different weight or in different positions on the table cause only negligible variations in the frequency response.

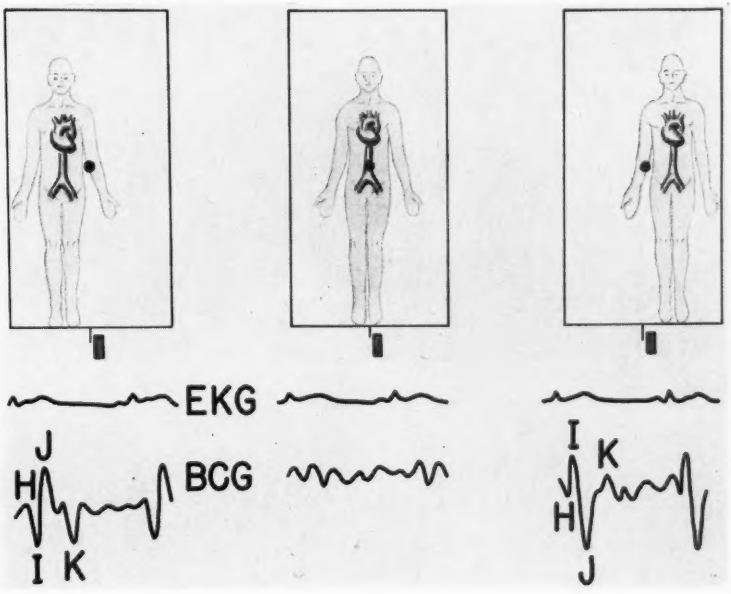

Fig. 2.—Torsion ballistocardiogram of a patient in various positions on the ballistocardiograph table. In the position on the left-hand side, an upper recoil produces a clockwise motion of the table; the same motion in the position in the middle produces no motion of the table; and in the position on the right-hand side of the drawing, an upward force causes a counterclockwise rotation. Note how the H wave is reversed (as well as the other wave) in direction as the patient changes position in relation to the center of rotation. Transverse forces would produce no appreciable changes in either the left- or right-hand positions on the table. The positions on the left and right sides of the table emphasize head-foot forces. The normal ballistocardiogram is similar to the one on the left-hand side of the table.

Standardization of the ballistocardiogram is done by applying a known force to the table end with the patient lying on the table in the selected position. To accomplish this, a mass of 1,500 Gm. was attached by a spring to the table top and caused to oscillate electrically. The recorded amplitude of these sinusoidal table oscillations provides the system calibration.

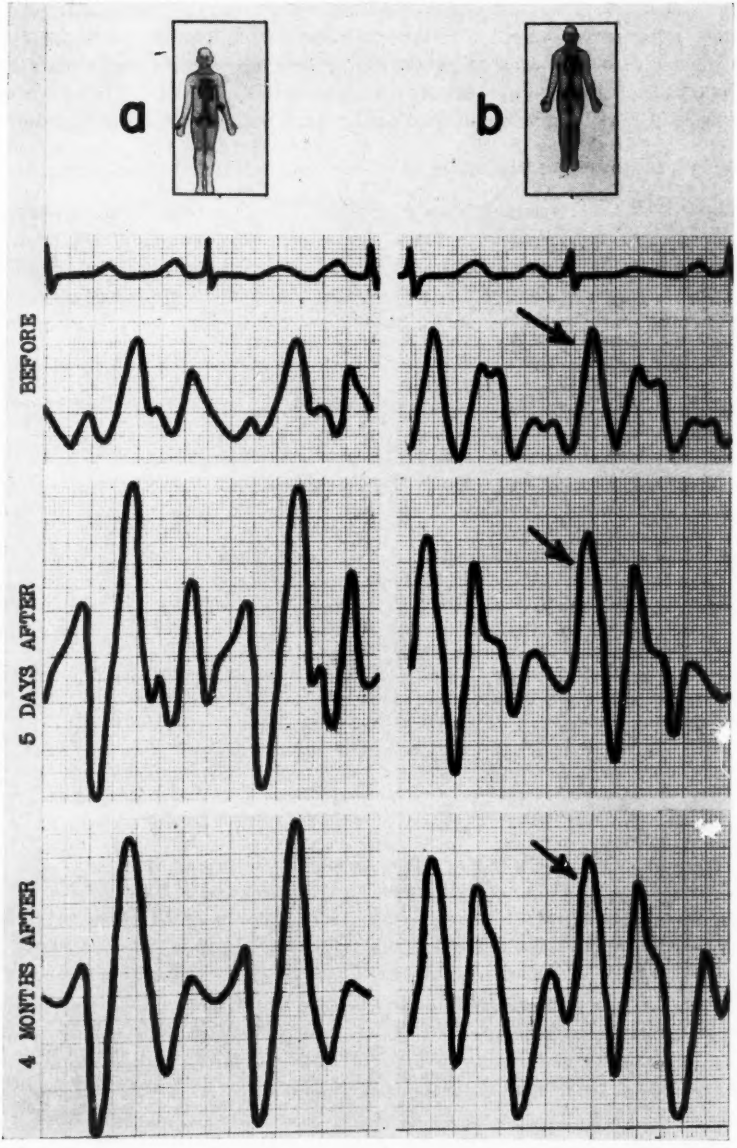

Fig. 3.—Mitral stenosis. Note the comparative increase in the amplitude of the diastolic complexes, particularly with the patient in the position where longitudinal vectors are emphasized (*a*). In the other position (*b*) a prominent wave in early systole (as indicated by the arrow) is seen. The ventricular complex becomes larger following operation; the abnormal wave believed to represent movement of the enlarged left auricle persists.

All records were taken in mid-respiration with the patient lying supine. An upward peak in the ballistocardiogram represents a clockwise rotation of the table top (viewed from above), a downward swing, a counterclockwise rotation. It must be recalled that this is the direction of the recoil force rather than the actual acceleration of the blood, which is directly opposite to the recoil.

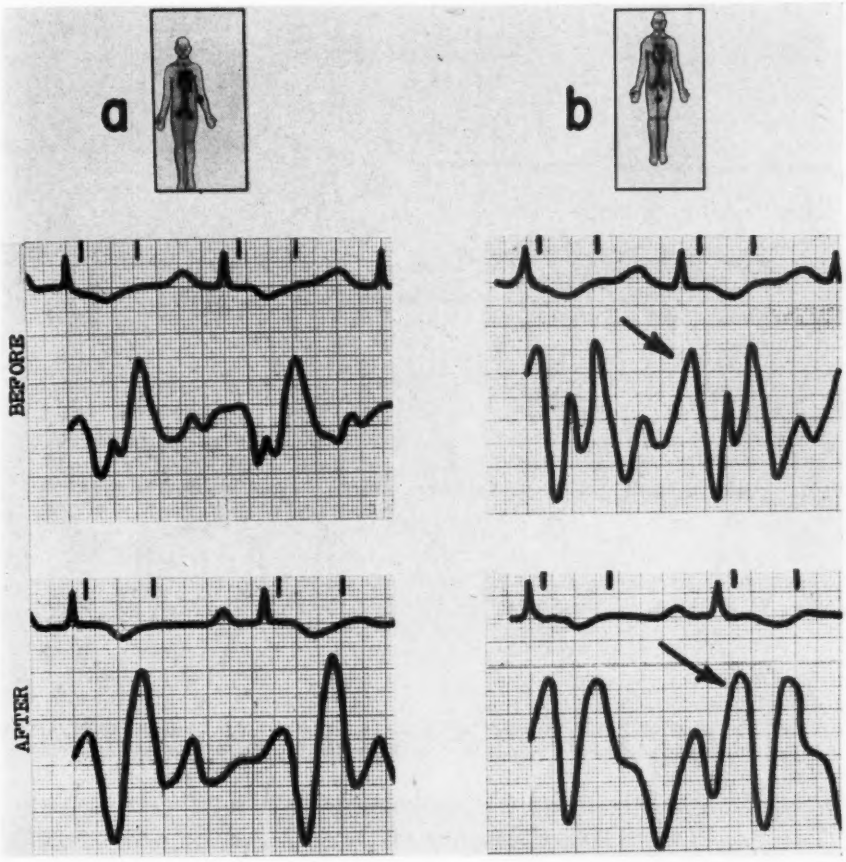

Fig. 4.—Mitral stenosis. Changes similar to those seen in Fig. 3 are observed.

Simultaneous electrocardiograms and/or phonocardiograms were taken to serve as a time reference only.

RESULTS

A. The Normal Ballistocardiogram.—Immediately following systolic activity, as denoted by the *R* wave of the electrocardiogram or the last portion of the first heart sound, is a deflection in the ballistocardiogram termed the

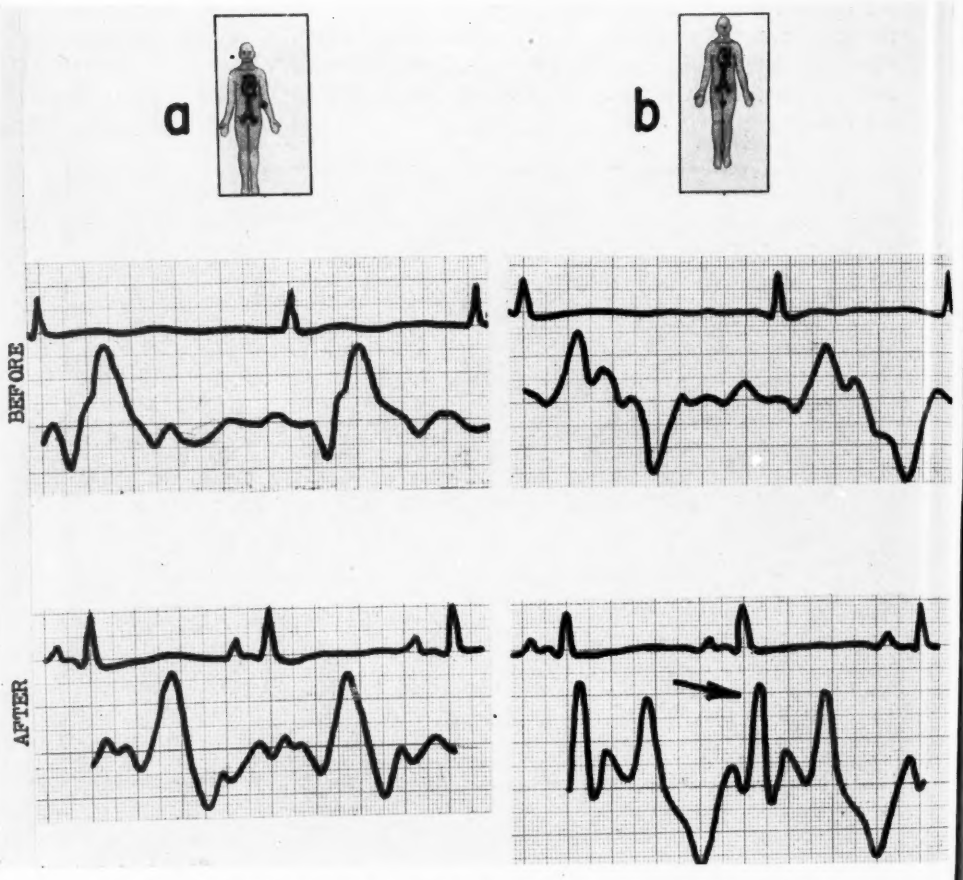

Fig. 5.—Mitral stenosis. Auricular fibrillation preoperatively, normal sinus rhythm postoperatively. Note that the abnormal wave in early systole (see arrow) is present only during normal sinus rhythm.

H wave. This subsequently reverses its direction as the *I* wave, generally deviating below the original base line and occurring roughly simultaneously with the onset of the femoral pulse and slightly before the peak of the carotid pulse wave. The *J* wave is much greater in amplitude and is related temporally to the ascending slope of the femoral pulse and the peaks of the carotid and abdominal aortic pulsations. The last major wave of the systolic complex, the *K* wave, is large and is almost coincident with the second heart sound, the descending portion of the femoral pulse, and the dicrotic notch of the carotid pulse wave. Subsequent other waves, designated as *L*, *M*, and *N*, appear to be related to activities during diastole and are not as well defined as the *H*, *I*, *J*, and *K* systolic complexes.[16]

The normal torsion ballistocardiogram and some of the alterations produced by changing the patient's position on the table are illustrated in Fig. 2. Here it may be noted that an upward wave on one side of the center of rotation becomes a downward deflection on the other side, demonstrating use of the torsion ballistocardiography in determining the site of origin of wave forms.

Fig. 6.—Acute experimental coarctation produced by progressive occlusion of the thoracic aorta in the dog. Ballistocardiograms taken to emphasize longitudinal vectors. In *A* the normal tracing is shown; in *B* approximately one-third of the lumen is occluded with bowing and deforming of the K wave; in *C* the aorta is almost completely obstructed with notching of the K complex. Reflex slowing of the heart is noted, a compensatory mechanism for the acute aortic obstruction.

B. Mitral Stenosis.—Examination of tracings obtained from patients with primarily mitral valvular disease revealed abnormal records with fairly uniform qualitative changes (Figs. 3 to 5). It might be anticipated that these lesions would influence the ballistocardiogram by virtue of the altered stroke volume. A marked diminution in the entire ventricular complex was found with comparative increase in the diastolic components.[17, 18] This is illustrated by the small abnormal I and J waves and their delayed onset. Following valvulotomy these waves increased markedly in amplitude, no delay was seen in their onset, their configuration was normal, and the diastolic waves became proportionally smaller. Indeed in those cases where a good clinical result was obtained, the ballistocardiogram following operation was interpreted as being

essentially normal. These changes were found in the ballistocardiogram taken in a position where the head-foot forces were accentuated (Figs. 3, *a*, 4, *a*, and 5, *a*).

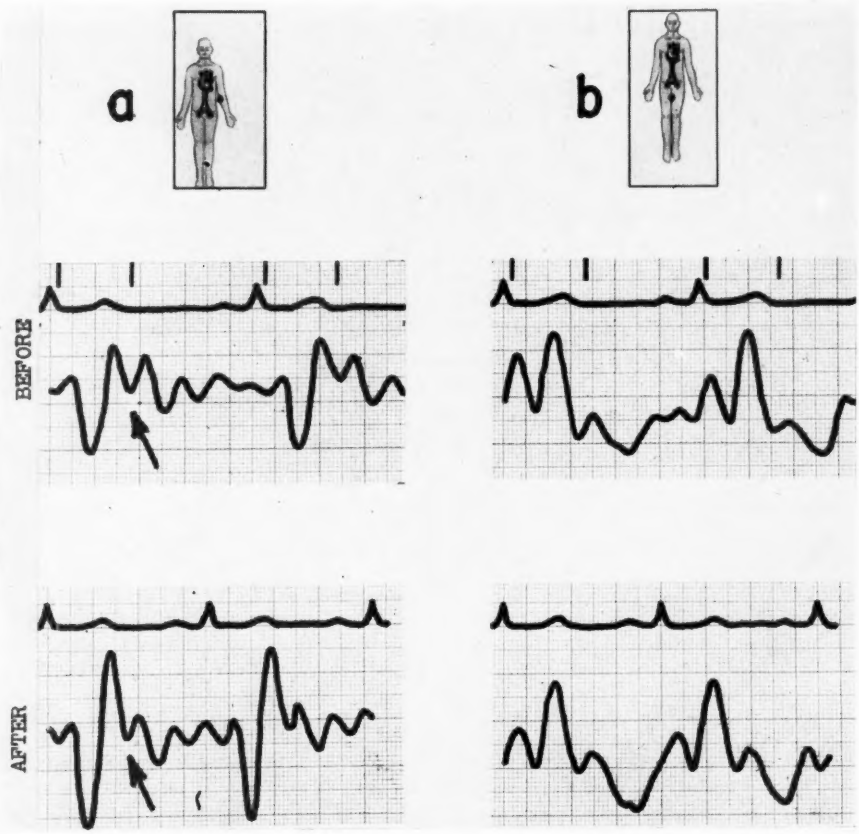

Fig. 7.—Coarctation of the aorta. Note the deformed wave (see arrow) and its return toward normal following operation. The I:J ratio is greater postoperatively. These abnormalities are best seen in *a* where longitudinal vectors are accentuated.

With the patient placed in a position (longitudinal axis of the table through its center) to emphasize transverse vectors, a prominent wave synchronous or slightly after the R wave of the electrocardiogram was noted (Figs. 3, *b*, 4, *b*, and 5, *b*). This remained following valvulotomy. Its direction indicated a right-to-left reactive force. In those patients with mitral stenosis who were fibrillating this wave was not found. However, in one patient who was fibrillating initially (Fig. 5, *a*) and reverted to a normal sinus rhythm postoperatively, the wave was noted, but only during normal rhythm (Fig. 5, *b*). Because of its indicated site of origin and its occurrence during normal

sinus rhythm solely, it is believed that this wave emanates from the enlarged left auricle; it probably represents movement of the auricle rather than the blood moved by contraction of the auricle.

No essential differences in ballistocardiographic patterns were found between those patients with mitral stenosis and mitral insufficiency, or their combination. Most of the patients undergoing mitral valvulotomy had cardiac catheterization preoperatively and postoperatively, but only a crude correlation was found between the calculated stroke volume and the amplitude of the ventricular complex of the ballistocardiogram.

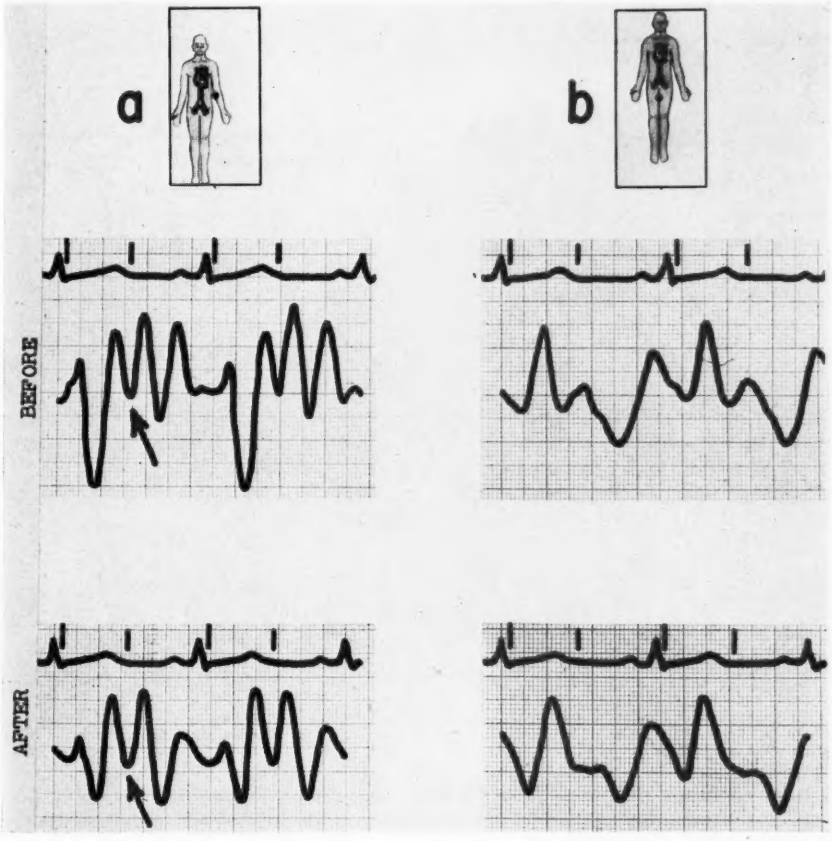

Fig. 8.—Coarctation of the aorta; similar changes as described in Fig. 7, but abnormal ballistocardiogram still present following operation. The coarctation could be repaired only by insertion of a homologous aortic graft.

C. Coarctation of the Aorta.—That a distinctive pattern of ballistocardiography is obtained in the patient with coarctation has been appreciated for many years.[5, 19-21] Abnormalities in aortic flow are indicated chiefly by major

changes in the K wave and minor abnormalities in other complexes. These deviations are well demonstrated in Fig. 6 which depicts the malforming of the K wave caused by an increasing obstruction to the thoracic aorta in a dog.[22]

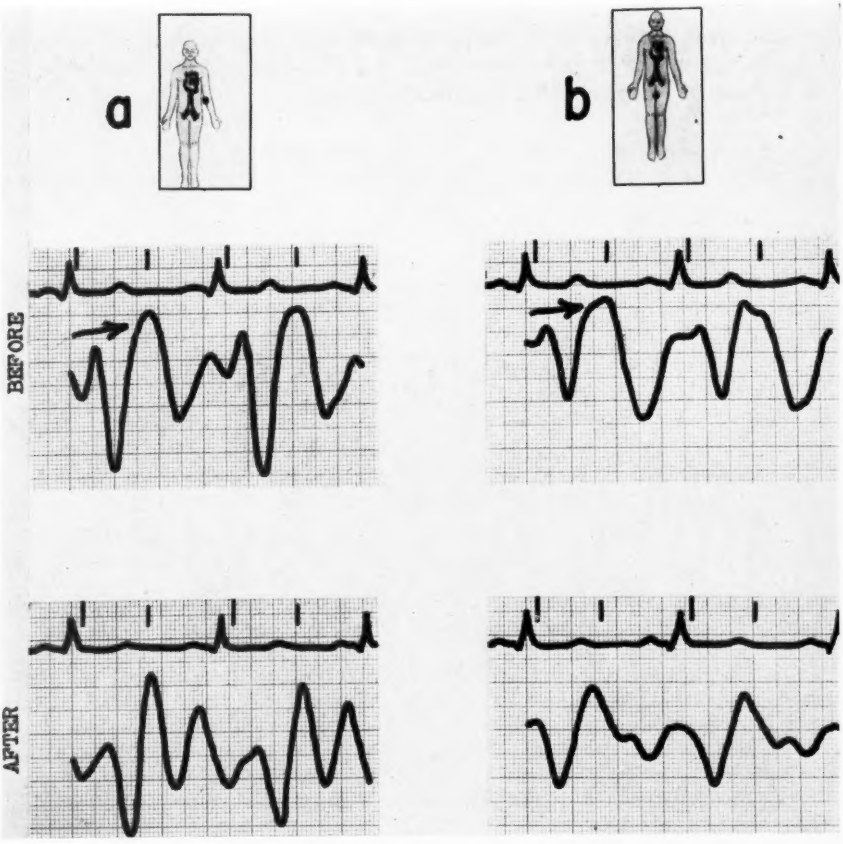

Fig. 9.—Persistent ductus arteriosus. Before operation an abnormal wave (see arrow) is seen in late systole, particularly in *b* where transverse vectors are accentuated. This abnormality disappears after operation.

Coaretation of the aorta is perhaps the best example where ballistocardiography can be used as a diagnostic procedure although even in this condition it is of relatively little practical importance. The changes in the ballistocardiogram in coaretation of the aorta are in the latter part of the ventricular complex and are due to the abrupt narrowing of the aorta with its marked impeding of the blood flow. The alterations are due to a greater than normal flow headward during the first half of systole producing an I wave of longer duration and greater amplitude than normal and a delayed and less accelerated

flow footward with a smaller and often delayed J wave with the absence or diminution of the K wave. These abnormalities were observed in all our patients with coarctation and were found to be reversed in varying degrees after operation (Figs. 7 and 8). The normal ratio of the I:J amplitudes is about

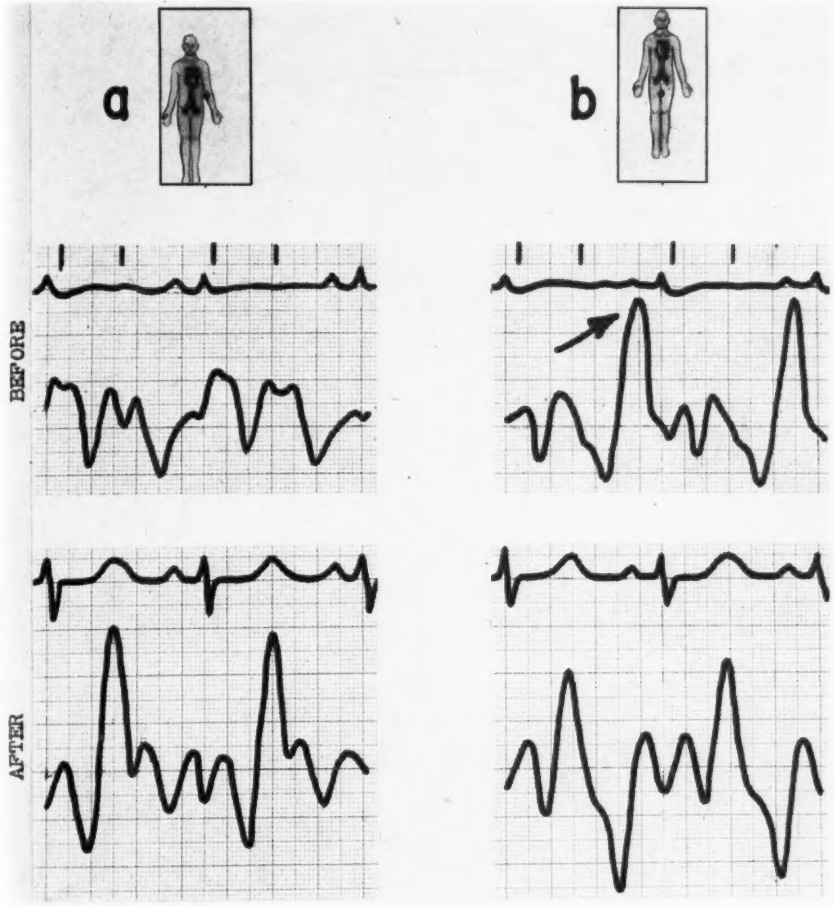

Fig. 10.—Chronic constrictive pericarditis. Attention is directed to the large wave occurring in diastole in the position where transverse vectors are emphasized (see arrow). This disappears after operation as the ballistocardiogram becomes essentially normal.

1:1.2, but in those patients with coarctation it is decreased, averaging about 1:0.64 in our series. The ratio following operation was found to be about 1:1. The ballistocardiogram thus provides an indication of the amount of blood passing through the aorta and may be of value in assessing this group of patients postoperatively.

D. Persistent Ductus Arteriosus.—It would be expected that in patients with an appreciable amount of blood flow through a ductus arteriosus, a vector could be ascertained pertinent to the ductus. Such a wave was noted in the ballistocardiograms of our patients with ductus arteriosus. Normally the J wave is terminated at the end of systole and returns to the base line, but in

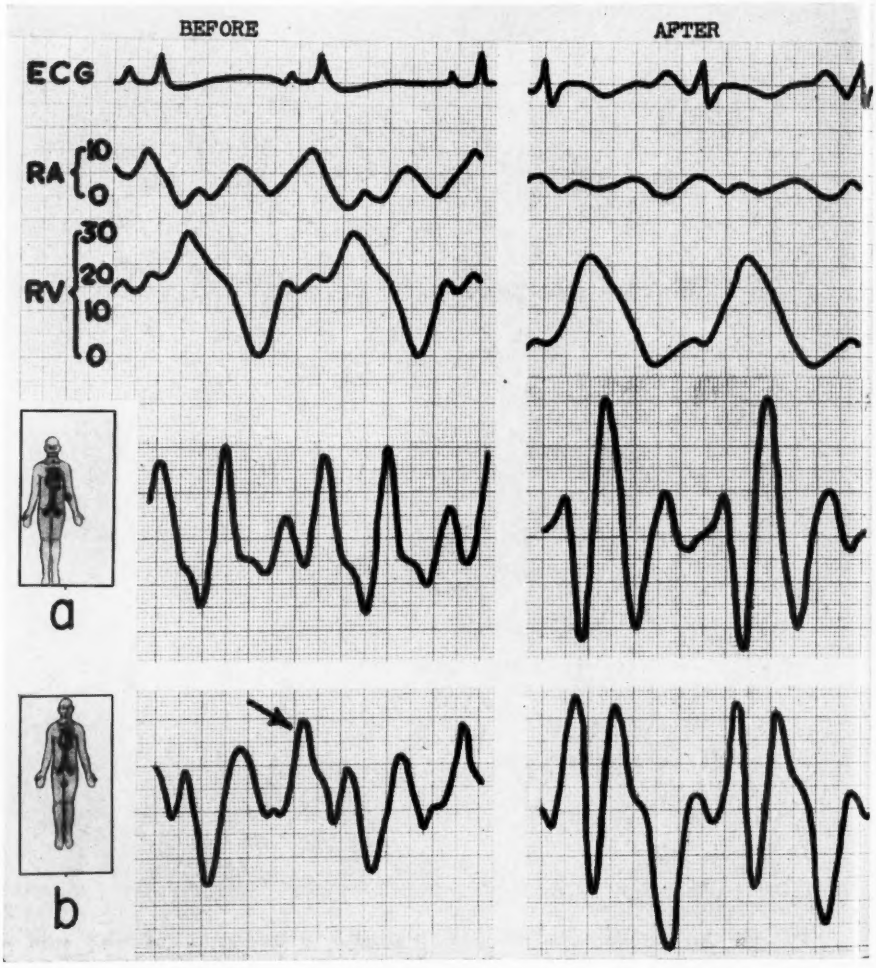

Fig. 11.—Chronic constrictive pericarditis. Changes similar to those mentioned in Fig. 10 correlated with right auricular and right ventricular pressure changes.

these patients a strong positive wave persisted. This was particularly accentuated when the patient was in the transverse position (Fig. 9, *b*). Since the wave deviated upward and was most prominent with the patient so placed, it must be a vector acting from right to left with a headward component.

This agrees perfectly with the recoil expected from blood moving through the ductus arteriosus. In all cases this wave complex disappeared following operation (Fig. 9).

E. Chronic Constrictive Pericarditis.—This condition almost invariably produces an abnormal ballistocardiogram,[23, 24] the record being characterized by unusually large diastolic waves, occupying most of diastole, and occasionally extending into early systole. Often these waves are larger than the systolic complexes. It has been noted previously that these were particularly prominent in the lateral vector projection, rather than the conventional head-foot position, and our observations confirm this. The abnormal ballistic forms are attributed to the more rapid surge of blood into the ventricle during diastole, this being secondary to the elevated venous pressure, and the abrupt increase in the interventricular pressure because of the inelasticity and rigidity of the ventricular walls. The accentuation of these abnormal complexes by placing the patient in a position to emphasize the transverse vectors is shown in Figs. 10, *b*, and 11, *b*.

Following pericardiectomy there is a gradual reversion of the abnormal ballistocardiogram to a normal one, although this may not occur for several months.

SUMMARY

1. Torsion ballistocardiography is briefly described, and its utilization in analysis of vectors comprising the ballistocardiogram is discussed.

2. Abnormal ballistic complexes occurring in patients with mitral stenosis, coarctation of the aorta, ductus arteriosus, and chronic constrictive pericarditis, and their changes following operation are illustrated.

3. The origin and factors causing the abnormal ballistocardiograms in these groups of cardiovascular diseases are speculatively reviewed.

REFERENCES

1. Gordon, J. W.: On Certain Molar Movements of the Human Body Produced by the Circulation of Blood, J. Anat. and Physiol. **11**: 533, 1877.
2. Starr, I., Rawson, A. J., Schroeder, H. A., and Joseph, N. R.: Studies in the Estimation of Cardiac Output in Man and Abnormalities in Cardiac Function From the Heart's Recoil and the Blood's Impact, Am. J. Physiol. **127**: 1, 1939.
3. Nickerson, J. L., and Curtis, H. J.: The Design of the Ballistocardiograph, Am. J. Physiol. **142**: 1, 1944.
4. Dock, W., and Taubman, F.: Some Technics for Recording the Ballistocardiogram Directly from the Body, Am. J. Med. **7**: 751, 1949.
5. Hamilton, W. F., Dow, P., and Remington, J. W.: The Relationship Between the Cardiac Ejection Curve and the Ballistocardiographic Forces, Am. J. Physiol. **144**: 557, 1945.
6. Scarborough, W. R., Beser, J., Talbot, S. A., Mason, R. E., Singewald, M. L., and Baker, B., Jr.: A Method for Recording Ballistocardiographic Vectors, Bull. Johns Hopkins Hosp. **87**: 235, 1950.
7. Scarborough, W. R., Davis, F. W., Jr., Baker, B. M., Jr., Mason, R. E., and Singewold, M. L.: A Review of Ballistocardiography, Am. Heart J. **44**: 910, 1952.
8. Gubner, R. S., Rodstein, M., and Ungerleider, H. E.: Ballistocardiography. An Appraisal of Technic, Physiologic Principles, and Clinical Value, Circulation **7**: 268, 1953.
9. Alexander, R. S.: Factors Determining Contour of Pressure Pulses Recorded From the Aorta, Federation Proc. **11**: 738, 1952.
10. Remington, J. W.: Volume Determination of Aortic Pulse, Federation Proc. **11**: 750, 1952.

11. Peterson, L. H.: Certain Physical Characteristics of Cardiovascular System and Their Significance in the Problem of Calculating Stroke Volume From Arterial Pulse, Federation Proc. **11**: 762, 1952.
12. Opdyke, D. F.: Genesis of Pressure Pulse Contour Method for Calculating Cardiac Stroke Index, Federation Proc. **11**: 733, 1952.
13. Huggins, R. A., and Smith, E. L.: Validity of Pulse Contour Method for Determining Cardiac Output, Federation Proc. **11**: 767, 1952.
14. Ernsthausen, W., Von Wittern, W., and Reissmann, K.: The Torsion Ballistocardiograph, Memorandum report MCRexD-696-11 6A, June 29, 1948. U.S.A.F., Air Material Command, Wright Patterson Air Force Base, Dayton, Ohio.
15. Reissmann, K. R., and Dimond, E. G.: Torsion Ballistocardiography With Special Reference to Patterns in Surgically Amenable Cardiovascular Diseases, Circulation. (In press.)
16. Thompson, W. B., Rappaport, M. B., and Sprague, H. B.: Ballistocardiography. II. The Normal Ballistocardiogram, Circulation **7**: 321, 1953.
17. Dock, W., Mandelbaum, H., and Mandelbaum, R. A.: Ballistocardiography in Medical Practice, J. A. M. A. **146**: 1884, 1951.
18. Gubner, R.: Selective Synchronous Recording of the Ballistocardiogram and Electrocardiogram on a Single Channel, Circulation **4**: 239, 1951.
19. Brown, H. R., Jr., Hoffman, M. J., and De Lalla, V., Jr.: Ballistocardiograms in Coaretation of the Aorta, New England J. Med. **240**: 715, 1949.
20. Nickerson, J. L., Humphreys, G. H., Deterling, R. A., Fleming, T. C., and Mathers, J. A. L.: Diagnosis of Coarctation of the Aorta With the Aid of the Low-Frequency, Critically-Damped Ballistocardiograph, Circulation **1**: 1032, 1950.
21. Murphy, R. A.: Ballistocardiographic Patterns in Intraluminal Aortic Obstructions, Am. Heart J. **39**: 174, 1950.
22. Smith, R. F.: The Effects of Acute, Partial, and Complete Obstruction of the Thoracic Aorta in the Dog, Master's Thesis, University of Kansas Medical Center, 1953.
23. Starr, I., and Schroeder, H. A.: Ballistocardiogram. II. Normal Standards, Abnormalities Commonly Found in Diseases of the Heart and Circulation, and Their Significance, J. Clin. Investigation **19**: 437, 1940.
24. Nickerson, J. L.: Some Observations on the Ballistocardiographic Pattern, With Special Reference to the H and K Waves, J. Clin. Investigation **28**: 369, 1949.
25. Scarborough, W. R., McKusick, V. A., and Baker, B. M., Jr.: The Ballistocardiogram in Constrictive Pericarditis; Before and After Pericardiectomy, Buil. Johns Hopkins Hosp. **90**: 43, 1952.

THE ROLE OF THE BRONCHIAL ARTERY CIRCULATION IN THE ETIOLOGY OF PULMONARY AND PERICARDIAL SUPPURATION

An Experimental Study

Richard S. Hahn, M.D.* (by invitation), Emile Holman, M.D.,** and John B. Frerichs, M.D.*** (by invitation)
San Francisco, Calif.

DESPITE the gratifying decrease in the mortality of pulmonary abscess and suppurative lesions of the pericardium during the past decade, their etiology remains obscure.[1-3]

Most students of pulmonary abscess still maintain that the predominant etiological factor is the aspiration of infected material and disclaim the possibility of septic vascular embolism. Many excellent clinical studies support this view,[4-6] but the experimental evidence is not so conclusive.[7-11] The many unsuccessful attempts to produce pulmonary abscesses by aspiration are in marked contrast to their almost invariable production following the introduction of a septic embolus into the jugular vein as reported first in 1926.[12-16] These studies further suggested that pulmonary infarction of considerable size may be a necessary precursor to the development of an abscess. Recent studies of Liebow, Ellis, Shedd, and their co-workers, supplementing the original observations of Mathes and others,[17-24] have shown that the hemorrhagic pulmonary infarction which follows massive embolism may actually be due to the destructive high-pressure inflow from the patent bronchial artery system. In animals in which the bronchial artery was successfully occluded, the production of massive embolism did not result in typical hemorrhagic infarction.

With these concepts in mind, a series of experiments were designed to embolize the bronchial artery with multiple septic particles, to determine, if possible, the role of this vascular system in infections of the lung and whether massive infarction is a necessary precursor to pulmonary abscess.

A few pilot experiments were performed by injecting in vivo the right posterior bronchial artery in the dog, after the method of Ellis, with a suspension of India ink (Fig. 1). The rapid appearance of the ink particles in the vessels supplying the pericardium and myocardium was most striking. This phenomenon was evident even in the preparations in which preliminary complete ligation of both right and left coronary arteries and the pericardiophrenic

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*Benjamin Swig Fellow in Surgery 1952-53, Stanford University School of Medicine.
**Professor of Surgery, Stanford University School of Medicine.
***Assistant Clinical Professor of Pathology, Stanford University School of Medicine, and Chief, Department of Pathological Anatomy, Veterans Administration Hospital.

arteries at their origin had been performed (Fig. 2). Many fine terminal branches of the bronchial artery could be seen accompanying the great vessels as they emerged through the pericardium. These hairlike vessels continued downward over the atrial walls, particularly on the right side and anastomosed freely with the right coronary artery. Numerous vessels could be seen spreading over the pericardium and anastomosing with branches of the pericardiophrenic artery, indicating the presence of direct anastomotic communications between these vascular systems. These observations suggested the possibility of a hematogenic dissemination of infection to both the lung and the heart by the same vascular system.

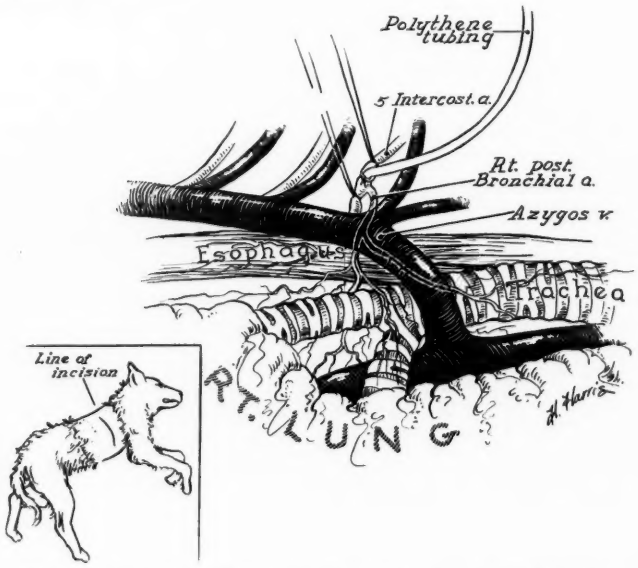

Fig. 1.—The right posterior bronchial artery in the dog as exposed through a right fifth intercostal incision. The parent intercostal artery has been ligated and cannulated preparatory to injection with septic emboli.

A review of the literature yielded little information concerning the extrapulmonary ramifications of the bronchial circulation. A casual mention of the bronchial artery supply to the mediastinal organs was made by Cauldwell and Anson with their co-workers,[25] Berry and Daly,[24] and Horine and Warner.[26] In 1932 Hudson, Mortiz, and Wearn[27] demonstrated filling of the bronchial artery by injections into the coronary circulations of the human heart. More recently, Tobin[28] has made brief reference to the bronchial arteries which supply the mid-portion of the esophagus, tracheobronchial lymph nodes, the vasa vasorum of the pulmonary artery, and the visceral pleurae.

METHOD

Adult mongrel dogs weighing 8 to 20 kilograms were used. Animals in which the presence of recent pulmonary disease was suspected were discarded. Veterinary Nembutal,

30 mg. per kilogram of body weight, and mechanical respiration were routinely employed. A standard right posterolateral thoracotomy was performed through the fifth intercostal space (Fig. 1). The right posterior bronchial artery was identified as it arose from the fifth, or sometimes from the fourth or sixth intercostal artery. The parent intercostal artery was ligated proximally and distally to the origin of the bronchial vessel; care was taken to avoid extensive dissection which would disrupt minute vessels. The azygos vein was not divided. The segment of intercostal artery between the ligatures was cannulated with polythene tubing, the inside diameter of which just snugly admitted a No. 20 gauge needle. A standardized quantity of septic material was injected, the cannula removed, and the vessel ligated permanently. Extreme care was taken to avoid gross contamination of the surrounding tissues; routine closure of the chest wall was performed. No drains or antibiotics were employed.

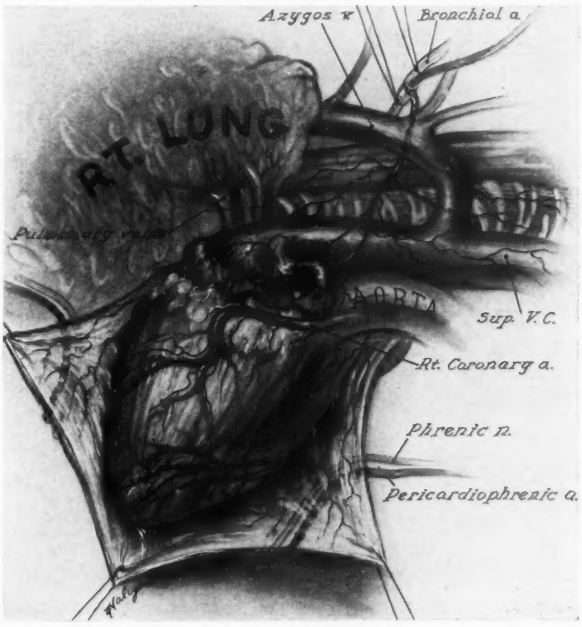

Fig. 2.—Diagrammatic reproduction of the extrapulmonary ramifications of the right posterior bronchial artery after India ink injection. Direct anastomotic communications with the right coronary and pericardiophrenic arteries are demonstrated.

Two series of animals were studied. Group I dogs received a standard 18- to 24-hour-old blood subculture inoculum of *Staphylococcus aureus*. The dosage was arbitrarily calculated on the basis of 0.5 c.c. per five kilogram of body weight. Fragments of fibrin and clot were employed as the embolic particles. The organism had been isolated from an infected posttonsillectomy fossa.

Group II dogs received the same organisms cultured in a glucose broth media. To this was added 1 c.c. of a freshly prepared suspension of barium sulfate, U.S.P. In a few dogs, Hill's* mass was employed. The dosage of this mixture was similarly calculated from body weight. Barium was used because it would provide a greater number of particles and also because its dissemination in the superficial vessels could be immediately visualized.

*Hill's mass is prepared from 200 Gm. of bismuth oxychloride, 100 Gm. of gum acacia, and 500 c.c. of distilled water.

RESULTS

Thirty animals were studied. These were equally divided into two groups.

Group I.—Of fifteen dogs in this series, all but two animals survived indefinitely without visible evidence of suppurative pulmonary disease. There was microscopic evidence of healed inflammatory processes in the visceral, parietal, and mediastinal pleurae. Minimal healed inflammatory lesions of the pericardium, esophagus, and tracheobronchial tree were observed. The parenchyma of the lung was essentially normal. No definite pulmonary abscess was found.

Fig. 3.—Photomicrograph of pericardium from animal in Group I demonstrating considerable thickening and evidence of acute inflammation throughout (hematoxylin and eosin, ×80).

Two animals died at five and six days, respectively, of acute cardiac tamponade, secondary to a purulent pericarditis with effusion. Grossly, there was empyema and diffuse cellulitis of the chest wall. All pleurae were intensely injected and the mediastinal space was filled with sanguinopurulent fluid. The esophagus, trachea, and major extrapulmonary bronchi bilaterally revealed evidence of acute transmural suppuration. The pericardium was tautly distended with sanguinopurulent fluid. It was grossly hemorrhagic and infected.

Microscopically, there was considerable thickening with acute and subacute inflammatory lesions throughout (Fig. 3). The myocardium was normal. The epicardium revealed a peripheral zone of acute inflammatory reaction. The lungs were normal with the exception of a visceral pleuritis. No gross or microscopic evidence of pulmonary infarction, suppuration, or abscess was found.

Group II.—Fifteen animals were in this series. The longest survival was one week. All of the remaining animals died within twenty-four to forty-eight hours. In thirteen animals the immediate cause of death was acute necrotizing myocarditis. Grossly, there was empyema

and cellulitis of the chest wall of a more severe degree than was observed in the two animals of Group I. The chest cavity was often filled with sanguinopurulent fluid as was the mediastinal space. An intense hemorrhagic inflammatory reaction of all pleurae, the esophagus, trachea and bronchi, and even the adventitia of the aorta was observed.

The pericardium was similarly involved and varying degrees of pericardial effusion were consistently encountered. In thirteen specimens, gross hemorrhagic zones were found chiefly in the right ventricle and atrium. Particles of barium were seen grossly in the right coronary artery (Fig. 4). The lesions varied from small, subendocardial to extensive transmural areas of involvement. In a few hearts, lesions of the left ventricle were seen posteriorly where intercoronary artery communications with the circumflex branch of the left coronary artery were present. No lesions of the area supplied by the left anterior descending artery were observed.

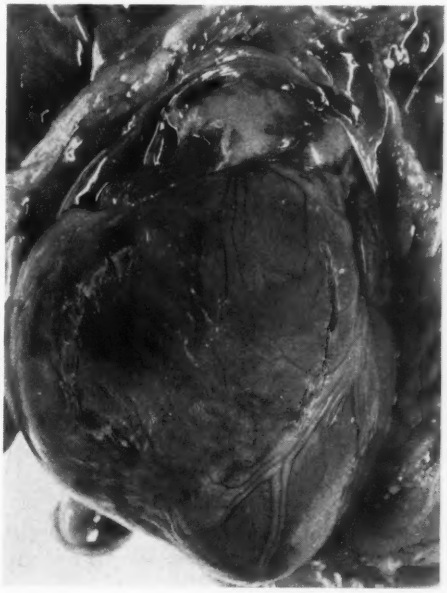

Fig. 4.—Typical heart from a Group II animal. The areas of right ventricular hemorrhage with barium particles in the right coronary arteries are demonstrated. A hemorrhagic lesion of the fatty tissue overlying the right atrium can be seen.

Microscopically, the lesions appeared to be typical of acute necrotizing myocarditis with interspersed areas of hemorrhage, acute inflammation, and destruction of the myofibrils (Fig. 5). Barium and staphylococcal emboli were frequently seen in the vessels of arteriolar size. The most intense inflammatory response was centered about the staphylococci. In several arterioles where there was relatively complete separation of the barium particles from the staphylococci, little or no inflammatory reaction appeared around the barium emboli. These findings substantiate the results obtained in two animals where barium was injected without organisms. The animals survived without evidence of hemorrhagic inflammatory vascular lesions. It would appear that the organisms and not the barium were responsible for the characteristic hemorrhagic pathology.

In contrast to the Group I animals, all but two dogs in Group II showed marked pulmonary parenchymal lesions (Fig. 6). Grossly, these appeared as bilateral areas of consolidation usually lobar in distribution. On incising the lung, whole blood poured forth.

Microscopically, the lesions appeared neither like that of acute consolidative pneumonitis nor hemorrhagic infarction (Fig. 7). The alveoli were filled with erythrocytes and a highly proteinic exudate containing little or no inflammatory elements. Closer inspection of the intramural branches of the bronchial arteries showed focal lesions of intense inflammation, often with rupture of the vessel wall which apparently accounted for the blood seen in the alveoli. Numerous emboli consisting of barium particles and staphylococcus organisms were seen within the lumen of the vessels. Again, no discrete pulmonary abscess, as such, was observed.

Fig. 5.—Photomicrograph of hemorrhagic myocardial lesion depicted grossly in Fig. 4. The epicardium is above, the endocardium below. Zones of hemorrhage are seen in the lower left with deeply staining clumps of staphylococci distributed among the myofibrils. Acute inflammatory elements surround most of the staphylococcal emboli (hematoxylin and eosin, ×17).

DISCUSSION

Experiments have been described in which an attempt was made to evaluate the role of the bronchial artery as a route for hematogenic infection of the lungs and pericardium. Under the conditions of the experiment, the use of small particles of infected blood clot in the animals Group I resulted uniformly in failure to produce pulmonary suppuration. These consistently negative results may be ascribed to the variable factors of host resistance, a too low virulence or inadequate dose of the organisms, or to an inadequate number of embolic particles employed.

It would appear that the higher incidence of pulmonary lesions observed in the animals in Group II can be attributed to the greater number of embolic particles which were successfully trapped, and not to the factors of resistance, dosage, and virulence of the organisms. Despite the more successful embolization of the bronchial artery circulation in Group II, no suppurative pulmonary

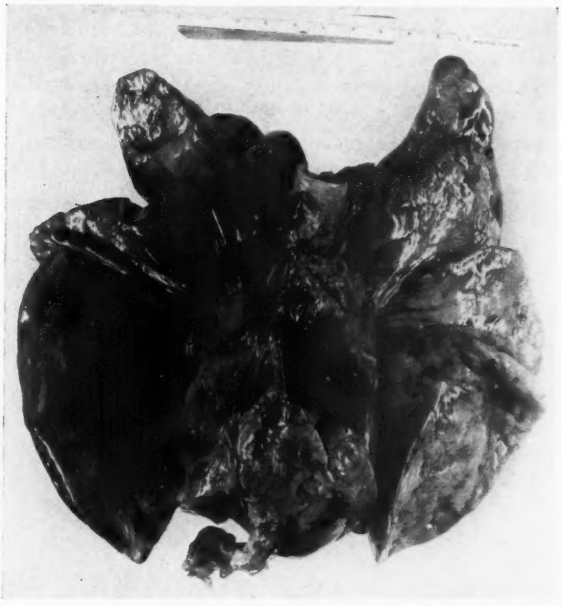

Fig. 6.—Gross specimen from typical Group II animal. There are marked hemorrhagic lesions involving the mediastinal pleura, trachea and pericardium. The right lung is dark, heavy, and nonair-containing as compared to the normal left lung.

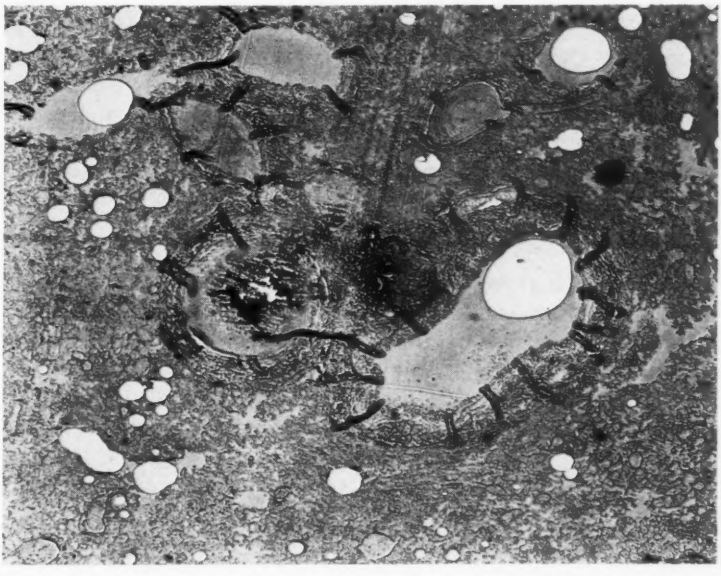

Fig. 7.—Photomicrograph of lung depicted in Fig. 6. The alveoli are filled with erythrocytes and a highly proteinic exudate. An intrapulmonary bronchus is seen to the left of center, a pulmonary artery on the right. A circumscribed lesion is seen within the wall of the bronchus consisting of staphylococci, barium particles, and polymorphonuclear leukocytes. There is an irregularity of one quadrant of the lesion suggesting rupture of its containing bronchial artery wall (hematoxylin and eosin, ×33).

lesions were observed. Likewise, no true pulmonary infarction was demonstrated in either series of animals. There is little doubt that the greater part of the bronchial artery blood flow was successfully occluded in Group II. The pulmonary arteries or veins did not contain barium emboli on microscopic examination.

The unexpectedly high incidence of myocardial and pericardial lesions was of great interest. The difference between the two animals in Group I which demonstrated primarily a pericardial involvement and the nine animals in Group II which uniformly presented additional myocardial lesions is again, we think, a matter of relative numbers of embolic particles employed. It is possible that embolic particles could have reached the myocardial capillary bed simply by direct shunting from bronchial arteries to pulmonary veins and then out the left heart. However, sections of tissues supplied by the systemic circulation have failed to reveal the presence of barium emboli. Moreover, it would seem to be unusual for the emboli to distribute so uniformly to the right ventricle and to avoid the left ventricle. The existence of these communicating anastomotic channels as demonstrated by the injection of India ink was demonstrable even when the entire coronary and pericardial arterial blood supply was completely ligated at its source. It is conceded, however, that more accurate methods of determining the exact route of dissemination should be studied further.

In summary, it would appear that the bronchial artery in the dog has a degree of ramification and collateralization hitherto not appreciated. Multiple septic emboli disseminated by this system did not produce definite pulmonary infarction or abscess. Indirectly, these results support the theory that pulmonary infarction is a necessary precursor to the pulmonary abscess produced by a vascular septic embolus. In view of the rapid death of Group II animals further studies are indicated. The high incidence of lesions of the mediastinal organs, pleurae, chest wall, heart, and pericardium suggest that the bronchial artery is more important as a mediator of infection to these structures than to the bronchopulmonary organs with which we usually associate this vascular system. The clinical importance of these observations deserves further study.

CONCLUSIONS

1. Multiple septic emboli injected into the right posterior bronchial artery of the dog failed to produce pulmonary infarction, suppuration, or abscess.

2. In contrast, there was a high incidence of suppurative lesions of the mediastinal organs, pleurae, chest wall, heart, and pericardium.

3. The anatomic communications between the bronchial and pericardial and intrinsic coronary artery systems have been demonstrated by India ink injection.

We are indebted to Miss Alice Violante and Mr. Dusan Surman for their kind assistance in this work.

REFERENCES

1. Smith, D. T.: Medical Treatment of Acute and Chronic Pulmonary Abscess, J. THORACIC SURG. **17**: 72-89, 1948.
2. Neuhof, H.: Acute Putrid Abscess of Lung, Surg., Gynec. & Obst. **80**: 351-354, 1945.

3. Brock, R. C.: Lung Abscess, 1952, Springfield, Ill., Charles C Thomas, Publisher.
4. Flick, J. B., Clerf, L. H., Funk, E. H., and Farrell, J. T., Jr.: Pulmonary Abscesses; Analysis of 172 Cases, Arch. Surg. **19:** 1292-1312, 1929.
5. Gwathney, J. T.: The Avoidance of Pulmonary Abscess With General Anesthesia in Nose and Throat Surgery, New York State J. Med. **22:** 394, 1922.
6. Cutler, E. L., and Gross, R. E.: Lung Abscess, J. THORACIC SURG. **6:** 125, 1936.
7. Aschner, P. W.: The Pathology of Lung Suppuration, Ann. Surg. **75:** 321, 1922.
8. Holman, E., Chandler, L. R., and Cooley, C. L.: Experimental Studies in Pulmonary Suppuration, Surg., Gynec. & Obst. **44:** 328-339, 1927.
9. Lambert, A., and Miller, J. A.: Abscess of Lung, Arch. Surg. **8:** 446, 1924.
10. Smith, D. T.: Experimental Aspiratory Abscess, Arch. Surg. **14:** 231-239, 1927.
11. Smith, D. T.: Fusospirochaetal Disease of the Lungs Produced With Cultures From Vincent's Angina, J. Infect. Dis. **46:** 303, 1930.
12. Holman, E., Weidlein, I. F., and Schlueter, S. A.: A Method for the Experimental Production of Lung Abscess, Proc. Soc. Exper. Biol. & Med. **23:** 266, 1926.
13. Holman, E.: The Etiology of the Postoperative Pulmonary Abscess, Ann. Surg. **83:** 240, 1926.
14. Holman, E.: The Postoperative Pulmonary Abscess, Northwest. Med. **25:** 290-294, 1926.
15. Holman, Emile, Chandler, L. R., and Cooley, C. L.: Experimental Studies in Pulmonary Suppuration, Surg., Gynec. & Obst. **44:** 328, 1927.
16. Holman, E., and Mathes, Mary E.: The Production of Intrapulmonary Suppuration by Secondary Infection of a Sterile Embolic Area, Arch. Surg. **19:** 1246-1261, 1929.
17. Liebow, A. A., Hales, M. R., Bloomer, W. E., Harrison, William, and Lindskog, G. E.: Studies in the Lung After Ligation of the Pulmonary Artery. II. Anatomical Changes, Am. J. Path. **26:** 177, 1950.
18. Shedd, D. P., Allev, R. D., and Lindskog, G. E.: Observations on the Hemodynamics of Bronchial Pulmonary Vascular Communications, J. THORACIC SURG. **22:** 537-548, 1951.
19. Ellis, F. H., Jr., Grindlay, J. H., and Edwards, J. E.: The Bronchial Arteries. I. Experimental Occlusion, Surgery **30:** 810-826, 1951.
20. Ellis, F. H., Grindlay, J. H., and Edwards, J. E.: The Bronchial Arteries. II. Their Role in Pulmonary Embolism and Infarction, Surgery **31:** 167-179, 1952.
21. Mathes, M. E., Reichert, F. L., and Holman, E.: An Experimental Method for the Radiographic Demonstration of the Bronchial and Pulmonary Arteries, Proc. Soc. Exper. Biol. & Med. **27:** 278-282, 1930.
22. Mathes, M. E., Holman, E., and Reichert, F. L.: A Study of the Bronchial, Pulmonary and Lymphatic Circulations of the Lung Under Various Pathologic Conditions Experimentally Produced, J. THORACIC SURG. **1:** 329, 1932.
23. Wood, D. A., and Miller, M.: The Role of the Dual Pulmonary Circulation in Various Pathologic Conditions of the Lungs, J. THORACIC SURG. **7:** 649, 1937-38.
24. Berry, J. L., and Daly, I. DeBurgh: The Relation Between the Pulmonary and Bronchial Vascular Systems, Proc. Roy. Soc. Med. **103:** 214-228, 1931.
25. Cauldwell, E. W., Dickert, R. G., Lininger, R. E., and Anson, B. J.: The Bronchial Arteries: An Anatomic Study of 150 Human Cadavers, Surg., Gynec. & Obst. **86:** 395-412, 1948.
26. Horine, C. F., and Warner, C. G.: Distribution of Pulmonary and Bronchial Circulation: Experimental Study, J. THORACIC SURG. **2:** 80-86, 1932.
27. Hudson, C. L., Moritz, A. R., and Wearn, J. T.: The Extra Cardiac Anastomosis of the Coronary Arteries, J. Exper. Med. **56:** 919-931, 1932.
28. Tobin, C. E.: The Bronchial Arteries and Their Connections With Other Vessels in the Human Lung, Surg., Gynec. & Obst. **95:** 741-750, 1952.

BRONCHOPULMONARY SARCOMA

LALLA IVERSON, M.D.*
WASHINGTON, D. C.

PRIMARY sarcoma of the lung is notably rare. If the diagnosis is limited to tumors composed of spindle-shaped cells of probable mesodermal origin, the majority of recorded cases can be eliminated. It is the purpose of this paper to consider the clinical and morphologic features characteristic of bronchopulmonary sarcoma and to discuss the differentiating criteria of its more frequent imitators.

REVIEW OF LITERATURE

In 1931, Ball[1] was able to collect only fifteen examples of pulmonary sarcoma from the European and American literature of the preceding thirty years. The majority of these were classified as "round-cell sarcomas," and only seven represented tumors composed of spindle-shaped cells.

The validity of cases reported since 1931 is equally difficult to determine. There is doubt as to the exact origin and histogenesis of some of the tumors. For example, four of the sarcomas occurred in women on whom hysterectomy had previously been performed.[2-5] Of these, only Cote's case, because of the location and morphologic features of the tumor, deserves serious consideration as a primary bronchopulmonary sarcoma. Likewise, pleura or mediastinum cannot be positively excluded as the site of origin of the tumor reported in other patients.[6-10] Many authors continue to report pulmonary sarcomas composed of round or poorly differentiated cells. These, according to inferences from the descriptions, may be variously interpreted as undifferentiated cell carcinomas or one of the types of lymphoma.[7, 11-19] Other omissions from the series are three cases in which the microscopic descriptions are incomplete[19-21] and three cases referred to in general articles.[22-24]

Thus, the medical literature from 1931 to 1950 contains only nine examples of spindle-cell sarcoma of probable pulmonary origin, raising the total of reported cases to sixteen for the period 1900 to 1950.

CASE REPORTS

In the material indexed at the Armed Forces Institute of Pathology as sarcoma of the lung are three additional examples which are similar, clinically and morphologically, to those selected from the medical literature. All three were intrapulmonary tumors composed of spindle cells associated with collagen or reticulum. Two of them came to clinical attention early in the course of the disease, when the patients were given routine x-ray examination. In all three cases the tumors were removed surgically, and in two the postoperative course has been uneventful in respect to recurrence or metastasis.

From the Armed Forces Institute of Pathology.
Received for publication Jan. 12, 1953.
*Present address: Department of Pathology, The University of the Philippines, College of Medicine, Manila, P. I.

Fig. 1.

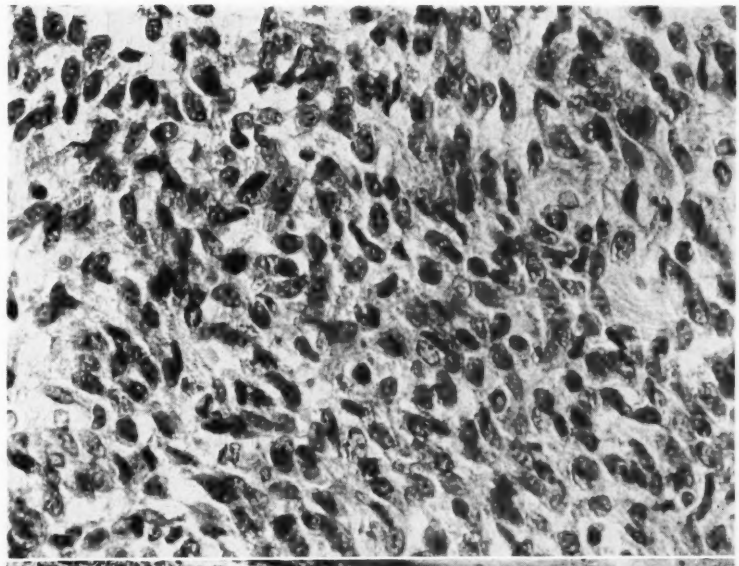

Fig. 2.

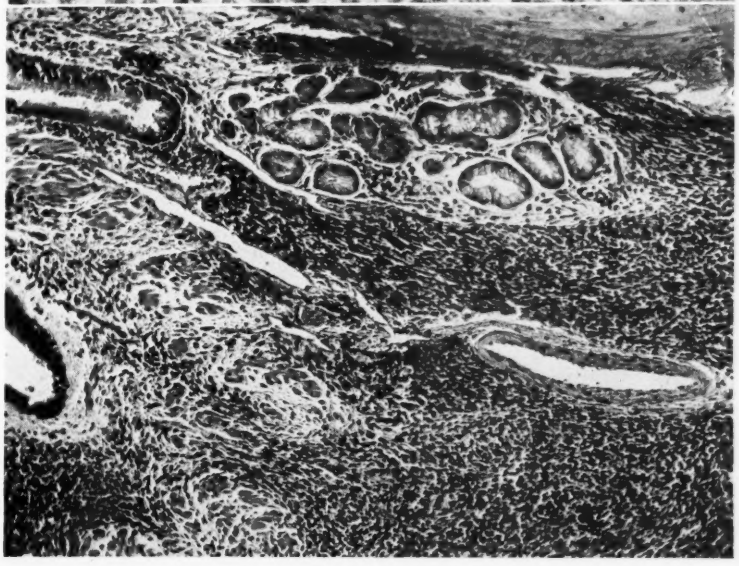

Fig. 1.—Intrapulmonary spindle-cell sarcoma; well-localized tumor composed of oval and spindle-shaped cells. Delicate fibrils associated with the tapering cytoplasmic poles. Hematoxylin and eosin, ×400. AFIP Acc. 161584.

Fig. 2.—Intrapulmonary spindle-cell sarcoma; this field resembles undifferentiated cell carcinoma of bronchus. Note tendency to sparing of glands and muscle bundles. Silver stains demonstrated reticulum. Hematoxylin and eosin, ×100. AFIP Acc. 161584.

CASE 1.—AFIP Acc. 161584. This 28-year-old white man had been without symptoms of any kind until his separation from the Army. Routine chest x-ray examination at that time revealed a rounded shadow in the upper lobe of the left lung. Bronchoscopic examination showed no abnormalities.

A firm, round mass was removed from the upper lobe of the left lung. On section, this proved to be a cyst measuring 4.5 by 3.5 cm., located near the hilum and containing yellow, gelatinous material. A secondary bronchus opened into the cyst and the bronchial mucosa was continuous with its smooth lining. The cyst wall was 2 mm. thick except at its lateral border where it merged with a nodular mass measuring 1 cm. in diameter. Microscopic sections showed the tumor to be composed of plump but elongated cells associated with reticulin formation (Fig. 1). There was local invasion of the wall of a small bronchus by the oval spindle-shaped cells (Fig. 2).

The patient's progress was followed until four years after lobectomy. He remained free from evidence of recurrence or metastasis of the tumor. Three years postoperatively there was enlargement of the glands of the right axilla, but biopsy revealed only chronic lymphadenitis.

CASE 2.—AFIP Acc. 210940. The patient was a 60-year-old white man. Although he had had no symptoms, an x-ray film of the chest taken during a general survey revealed a mass in the upper part of the right lung. Three months later, further x-ray examination confirmed these findings and a presumptive diagnosis of pulmonary cyst was made.

Right upper lobectomy was performed. The lung contained a solid globular mass, 9 by 8.5 cm. The tumor was well circumscribed and the pleura overlying it was smooth (Fig. 3). At one point the friable, grayish-white neoplastic tissue infiltrated the wall of a bronchus which was involved in the mass. Histologic preparations showed well-differentiated spindle-shaped cells with elongated reticulin-positive cellular fibrils (Fig. 4).

No metastasis was demonstrable at the time of operation, and three years later there was no evidence of recurrences.

CASE 3.—AFIP Acc. 271509. The patient was a 47-year-old white man. One year previously, at his annual physical examination, electrocardiograms and chest films had shown no abnormalities. Nine weeks prior to admission he complained of "tightness in the left chest" which interfered with deep respirations. At this time, x-ray examination of the chest revealed a lesion in the lower lobe of the left lung, and a diagnosis of pneumonia was made. The diminished breath sounds, decreased expansion, and inspiratory râles did not improve with therapy. Subsequent x-ray films demonstrated a mass, measuring 8 cm., in the suspected area, associated with elevation of the left leaf of the diaphragm. The mass rapidly increased in size during the first five weeks of hospitalization. The lung fields otherwise remained clear except for pleural thickening over the tumor.

Exploratory thoracotomy was performed two and one-half months after the onset of symptoms and a lobulated, grayish, friable tumor occupying the lower lobe of the left lung was removed. On gross examination no abnormalities of the bronchial tree and vascular system and no metastases were observed. Neither was there evidence of any possible primary site, other than the pulmonary parenchyma. X-ray therapy was administered postoperatively; it was again administered six months later, when the mass recurred. The patient's pain and dyspnea progressed until death, approximately one year after lobectomy. Post-mortem examination revealed tumor occupying the remainder of the left lung and a metastatic nodule in the lower lobe of the right. The original surgical specimen, like the metastatic nodules at autopsy, was composed of poorly differentiated spindle-shaped cells. The cells were associated with reticulin formation except in areas of necrosis, and more solid portions were marked by collagenous intercellular material (Figs. 5 and 6). There was local invasion at the periphery of the primary tumor (Fig. 7).

CHARACTERISTICS OF BRONCHOPULMONARY SARCOMA

These three cases, together with the sixteen examples selected from the medical literature from 1900 to 1950, are included in the total of 19 cases of

Fig. 3.

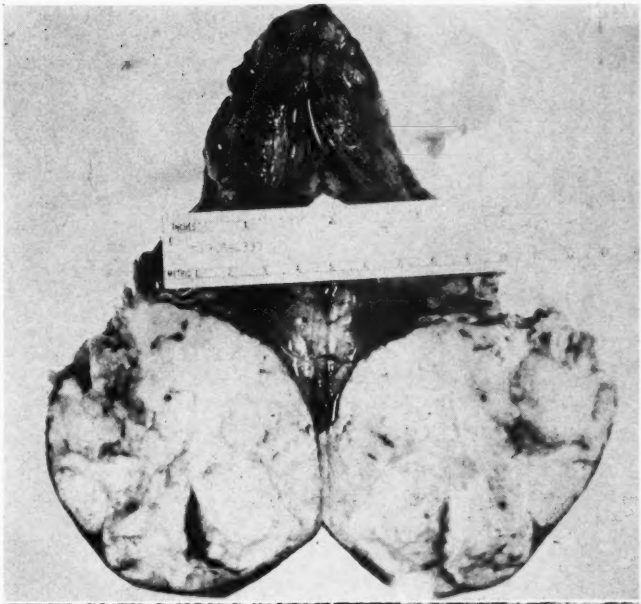

Fig. 4.

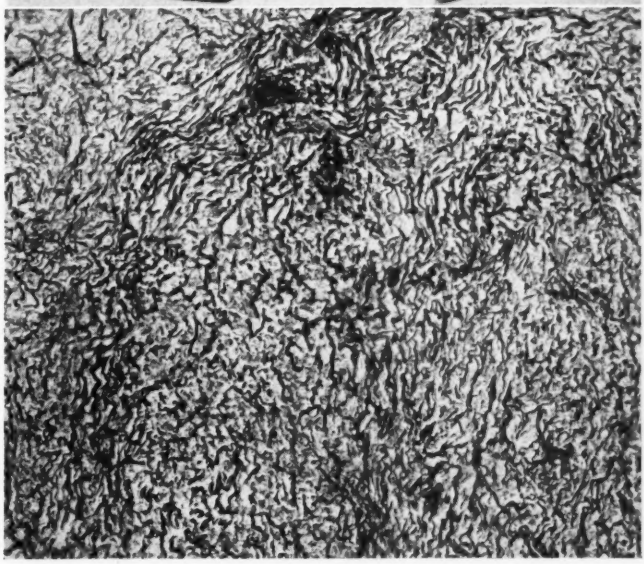

Fig. 3.—Intrapulmonary spindle-cell sarcoma (surgical specimen); a characteristic localized solitary primary tumor encroaching on bronchus. AFIP Acc. 210940.

Fig. 4.—Intrapulmonary sarcoma; reticulum formation helps to differentiate the tumor as sarcoma. Reticulum stain, ×235. AFIP Acc. 210940.

Fig. 5.

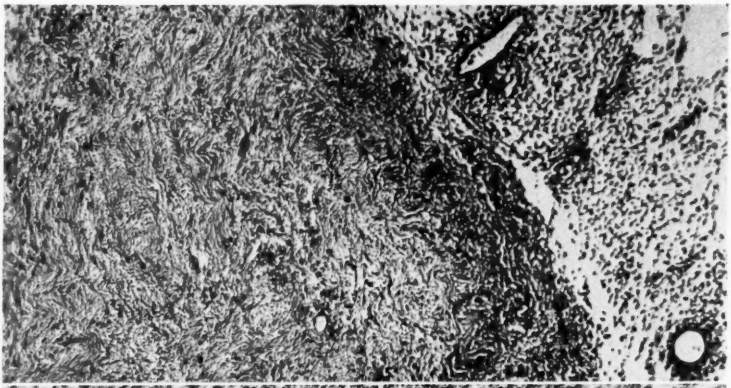

Fig. 6.

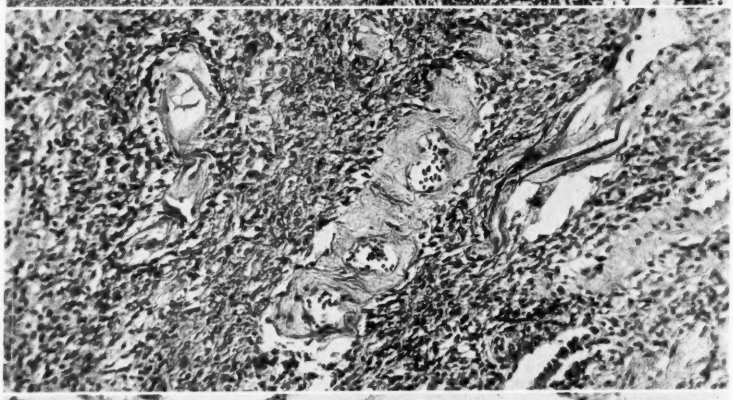

Fig. 7.

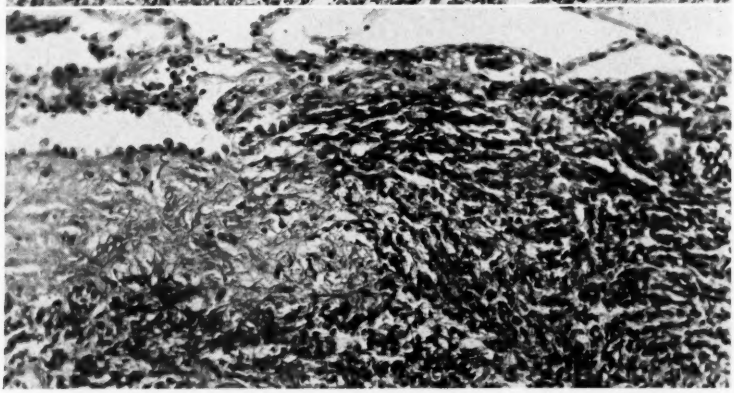

Fig. 5.—Intrapulmonary spindle-cell sarcoma; an area of collagenous fibers produced by the well-differentiated spindle-shaped cells borders a zone of necrosis where the tumor resembles reserve cell carcinoma. Masson stain, ×60. AFIP Acc. 271509.

Fig. 6.—Intrapulmonary spindle-cell sarcoma; in some areas the thick-walled vessels and collagen production suggest a benign tumor. Hematoxylin and eosin, ×100. AFIP Acc. 271509.

Fig. 7.—Intrapulmonary spindle-cell sarcoma; incomplete encapsulation of tumor resulting from expansile type of invasion by the poorly differentiated spindle-shaped cells. Hematoxylin and eosin, ×185. AFIP Acc. 271509.

intrapulmonary spindle-cell sarcoma summarized in Table I. It should be noted that this series, in order to include only those tumors of relatively certain pulmonary origin, necessarily represents a prejudiced sample of either early or premetastatic examples (Table I).

Morphologic Characteristics of Intrapulmonary Sarcoma.—Primary sarcoma of the lung is characteristically a solitary, firm, white tumor (Fig. 3) occurring in middle-aged persons. Sections from the tumors in the three cases reported here were compared with known sarcomas arising elsewhere and metastasizing to the lung. Attention was given to encapsulation, morphologic characteristics of the tumor cells, and extent of the tumor. Descriptions contained in the literature were also utilized in this study.

In the primary tumors the capsule, where intact, was dense and fibrous, but it was not always adequate to restrain the tumor which advanced through it and into the adjacent parenchyma in a broad, expansile fashion (Fig. 7). The degree of encapsulation tended to vary with the degree of cellular differentiation. Although some metastatic sarcomas were nodular, poorly encapsulated, and multiple, others demonstrated gross features identical with those of primary sarcoma.

In most of the microscopic sections the neoplastic cell was spindle-shaped or oval, often with the nucleus blunted at one pole and tapering at the other (Figs. 1, 2, and 7). Rare portions of these tumors bordering areas of hemorrhage and necrosis contained cells which in cross section appeared indistinguishable from lymphoid or "oat cells" except for the poorer chromatin content of their nuclei (Fig. 5). In these more cellular zones the tumor cells collected around thin-walled vessels. In all areas reticulin honeycombed the tumor, forming recesses for one or several cells, and was occasionally reinforced by varying amounts of other intercellular substance (Figs. 4 and 6). In the more compact portions of the tumor the cells tended to be more elongated and the reticulum more dense. Sometimes fine undulating fibrils, closely packed together, formed the background for extremely well-differentiated serpentine cells (Fig. 6).

Case 2 in this series is representative of a small number of other descriptions in the literature in which the neoplastic cell, as in leiomyosarcoma, is uniformly large and well-differentiated, possessing abundant cytoplasm. In these there were few mitoses. The cells were bundled together compactly and were intimately associated with tapering eosinophilic fibers staining for reticulum.

In summary, pulmonary sarcomas are composed of spindle-shaped cells productive of varying amounts of intercellular material, or cellular fibrils which may stain as collagen, reticulum, or smooth muscle. More often reticulin is the only positive-staining product.

Behavior of Intrapulmonary Spindle-Cell Sarcoma.—The peripheral location of spindle-cell sarcomas of the lung gives rise to such nonspecific symptoms as dyspnea, fever, or malaise. Thus, the clinical course is likely to be of long duration unless the lesion is an incidental discovery during routine examination. Because of its circumscription the tumor lends itself readily to removal by

TABLE I. INTRAPULMONARY "SPINDLE CELL" SARCOMA, CASES REPORTED 1900-1952

AUTHOR YEAR	AGE	SEX	DURATION	CLINICAL	GROSS RELATIONSHIP	EXTENSION OR METASTASES	MICROSCOPIC
Kobylinski[25] (Quoted by Ball) 1904	46	M	Asymptomatic		Left upper lobe Small firm tumor	None	Fibrosarcoma
Boschowsky[26] 1912	56	M	Not stated	Chest pain, cough, dyspnea	Left lung Large tumor, well-circumscribed, encapsulated	None	Spindle cells, varying degrees of differentiation
Stevens[27] 1912	24	M	4½ mo.	Dyspnea, pain, cough, swelling of rt. chest	Right lung Almost replaced by firm gray tumor	None	Spindle-cell sarcoma
Pritchard[28] 1921	23	F	16 mo.	Acute onset of fever, hemoptysis cough; 3½ cm. mass by x-ray; 10 mo. later 7 × 7¼ cm. At operation, 12 × 10 cm.	Right mid-lung Circumscribed, semisolid tumor, 800 Gm.	None	Spindle-cell sarcoma
Campatelli[29] 1926	53	F	30 mo.	Dyspnea, cough, vomiting	Right upper lobe Right mid-lung Firm, white tumor	None	Typical spindle-cell "fibrosarcoma"
Edwards[30] (Case 5) 1927	60	M	3 yr.	Hemoptysis, cough, recent back pain	Right lower lobe Firm, white lobulated tumor	None	"Plexiform sarcoma"
Ball[1] 1931	51	M	32 mo.	Cough, stuporous episodes, night sweats 7 mo. before death	Left lower lobe Firm, gray tumor, 20 × 20 × 13 cm.	None	Spindle cells with intercellular material; major portion poorly differentiated
Scheidegger[31] 1931-1932	49	F	7 mo.	Pleural effusion, dyspnea, edema; cystic mass, entire left lung	Left lower lobe Large, well-circumscribed tumor	None	Portions of tumor, dense, solid "like fibroma"; others "like sarcoma"; some giant cells
Mysh[32] 1933	38	M	2 yr.	Cough, dyspnea	Left upper lung Well-circumscribed tumor	(Surgical specimen) none	Spindle-cell sarcoma
Lyssunkin[33] 1935	48	F	2 yr.	History of tbc. cough, chest pain, signs cardiac failure for 2 yr.; mass rt. lung. Cachexia	Right lower lobe Well-circumscribed intrapulmonary tumor 20 × 16 × 11 cm. central portion firm and white; outer zone soft and necrotic; tuberculosis and bronchiectasis	None	Collagen-producing spindle cells; atypical mitoses; fat-containing cells in edematous peripheral portion
Johns and Sharpe[34] 1935	18	M	15 mo.	Cough, hemoptysis, sputum, loss of wt., 6 cm. density right lung enlarging and extending to pleura	Right lung Tumor occupying most of right lung, extending along wall of right main bronchus and protruding into lu-	Heart, via pulmonary vessels; opposite lung	Mitoses common; very cellular, spindle and round cells; silver stains show reticulum

Mass. General Hospital Case 22441[35] 1936	47	F	4 mo.	Pain, cough, dyspnea	Right lower and middle lobes Circumscribed, solid, lobulated mass	Small, solitary nodules on liver surface, kidney, spleen, opposite lung	Well-differentiated fibrosarcoma in primary tumor and spleen; multinucleated giant cells in secondary liver and lung nodules
Stohr and Sachs[36] 1937-1938	51	M	15 yr.† 6-7 yr.†	Joint pains, dyspnea, hypophyseal cyst	Right lower lobe Tumor "size of man's head"; well circumscribed	Pleura overlying tumor	Spindle cells; positive reticulum
Mass. General Hospital Case 24472[37]	63	M	1 mo.	Pain; operation 1 mo. after onset	Left lower lung Sharply defined, lobulated tumor within lung	(Surgical specimen) none	Fibrosarcoma of spindle cells
Ellis[38] 1939	51	M	2 yr.	Fever, cough, night sweats for 2 yr.; paralysis and convulsions for 1 mo.	Right middle lobe Soft friable, well-circumscribed tumor, 8 × 5 × 5 cm.; located toward periphery, 3 × 2 × 2 cm. mass at hilum protruding into bronchus	Brain; hilar node	Small interlacing spindle-shaped cells in dense sheets; very vascular; areas of necrosis; reticulum positive
Mass. General[39] Hospital Case 31271 1945	65	M	1 yr.	9 mo. weakness; pulmonary osteoarthropathy; died 3rd mo. P.O. with pneumonia and widespread skeletal metastases	Right lower lobe Large, round, well-encapsulated tumor; necrotic areas	Bone	Fibrosarcoma
AFIP NO. 161584	28	M	Asymptomatic	Round density by routine x-ray examination	Left upper lobe Cystic mass 4.5 × 3.5 cm. with 1 cm. nodule in cyst wall	None; 4 yr. P.O. living and well	Spindle-cell sarcoma with foci of intercellular collagen; reticulum formation
210940	60	M	Asymptomatic	Density discovered by mobile x-ray unit	Right upper lobe 9 × 8.5 cm., solid globular white tumor pleura intact; bronchus infiltrated in one area	None; 3 yr. later, patient living and well	Well-differentiated spindle-cell sarcoma
271509	47	M	2½ mo.	Tightness in chest; mass by x-ray. P.O. x-ray therapy. Mass recurred 6 mo. after operation. Death 14½ mo. after onset	At operation left lower lobe 5 cm. lobulated friable, grayish tumor. At autopsy: remainder of left lung involved	None at operation Right lower lobe at autopsy	Spindle-cell sarcoma producing reticulum and varying amounts of intercellular material

lobectomy or pneumonectomy. Analysis of Table I reveals that metastasis, recurrence, or extension occurred in only six of the nineteen examples; the absence of metastasis in the majority of remaining cases was demonstrated at autopsy after a clinical course averaging seventeen months.

These data indicate that intrapulmonary sarcoma is typical of other "low grade" sarcomas in that it is slow growing, tends to be well-circumscribed, and may effect no undue hardship on the patient if it is removed early and completely. It may, however, metastasize to bone or distant organs.

Although the infrequent metastasis or recurrence seems to assure a relatively good prognosis, sarcomas arising in the lung may be presumed to share the surprising potentiality for late metastasis observed in tumors of similar histologic types originating elsewhere, and it is presumed that further follow-up studies of the present series will elicit examples of unusual metastasis years after the removal of the primary tumor.

DISCUSSION

The clinical and anatomic data presented offer criteria by which pulmonary spindle-cell sarcoma may be defined. There are, however, other infrequently occurring tumors which are of importance to clinicians and pathologists in their combined approach to the diagnosis of bronchopulmonary sarcoma. These are (1) endobronchial sarcoma; (2) miscellaneous rare types of sarcoma such as chondrosarcoma, rhabdomyosarcoma, and liposarcoma; and (3) lesions that may imitate primary sarcoma of the lung.

Endobronchial Sarcomas.—Occasionally a sarcoma may arise in one of the main bronchi, there producing endobronchial obstruction (Fig. 8). This may be distinguished from sarcoma arising in the parenchyma not only by the symptoms resulting from its peculiar location but also by apparent intrinsic differences in the neoplasm itself.

In 1950, Black[40] collected from the literature at least four cases which he designated fibrosarcoma of the bronchus, and recorded another example. Three additional cases were reported by other authors to bring the total to eight.[41-43] These are summarized as "endobronchial spindle-cell sarcoma" in Table II.

The descriptions in Table II and study of sections from Cases 5 and 7 indicate that the cell type characteristic of these polypoid endobronchial tumors is large and plump with an oval vesicular nucleus (Fig. 9). The cells, usually better differentiated than those forming the majority of parenchymal sarcomas, may be blunt, tapered, or prolonged into fibrils which are silver-positive when stained for reticulum.

Although the exposed surface lends itself readily to biopsy, the diagnosis is made exceedingly difficult by these well-differentiated mesodermal neoplastic cells which, in the presence of secondary inflammation, may easily be mistaken for reticulo-endothelial cells or organizing fibroblasts (Figs. 10 and 11). Only when the entire tumor is studied following pneumonectomy or at autopsy does the neoplastic nature of the cell seem to be convincingly demonstrated.

A review of the clinical behavior of these "endobronchial sarcomas" affords further indication for separating them from pulmonary spindle-cell sarcomas.

Fig. 8.

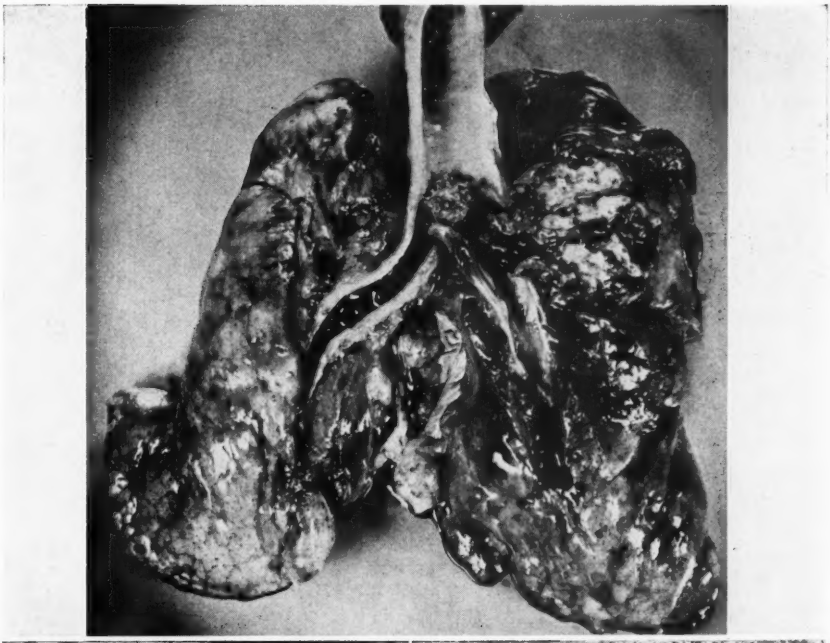

Fig. 9.

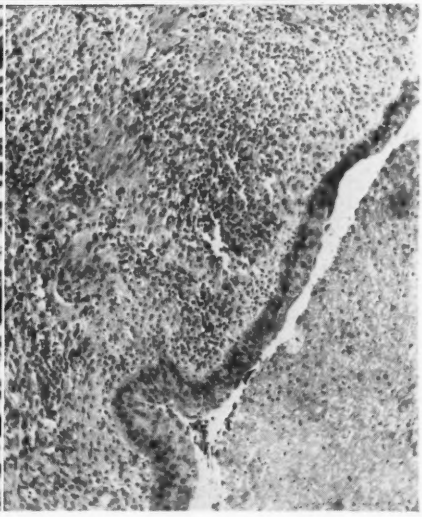

Fig. 10.

Fig. 8.—Endobronchial sarcoma (autopsy specimen); polypoid mass producing bronchial obstruction. AFIP Acc. 166844.

Fig. 9.—Endobronchial sarcoma: sections deep in the mass reveal neoplastic cells growing in a sarcomatous pattern. Hematoxylin and eosin ×450. AFIP Acc. 166844.

Fig. 10.—Endobronchial sarcoma; necrotic tumor obstructing the bronchus. Neoplastic nature of the lesion masked by the secondary inflammatory reaction. Hematoxylin and eosin, ×90. AFIP Acc. 166844.

TABLE II. ENDOBRONCHIAL SPINDLE-CELL SARCOMA CASE REPORTS

AUTHOR YEAR	AGE AND SEX	DURATION OF SYMPTOMS	CLINICAL SYMPTOMS	LOCATION OF TUMOR	GROSS RELATIONSHIP	METASTASES	MICROSCOPIC DESCRIPTION	DEATH OR DURATION
Baum[44] 1938	29 F	1½ mo.	Cough, chest pain, weight loss	Right main bronchus	Soft polypoid mass removed by bronchoscopy; x-ray therapy	None	Fibrosarcoma	Died of tbc. 2 yr. 9 mo. after onset. Squamous metaplasia of bronchus; fibrosis at biopsy site; no tumor remaining
Mass. General Hospital[45] 24202 1938	13 F	2 mo.	Pleuritic pain, weight loss, cough	Right main bronchus	Mass 1 × 1.5 cm. removed by bronchoscopy; thoracotomy attempted	None	Fibrosarcoma "growing at moderate rate" invading bronchial wall	Died of hemorrhage during operation
Pollack[46] 1939	19 M	7 mo.	Cough, pain, fever, weight loss, wheezing	Left main bronchus	Soft mass 2 × 3 cm. treated by bronchoscopy	None	Fibrosarcoma bronchus	No recurrence after 3 yr.
Lewis[47] 1946	14 M	1 mo.	Dyspnea, cyanosis, pain, cough	Orifice of right main bronchus up to coryna	Soft growth treated by bronchoscopy and x-ray	None	Fibrosarcoma with whorl arrangement and considerable pleomorphism; rebiopsy 3 mo. later showed more variation	X-ray normal 2½ yr. after onset
Randall and Blades[41] AFIP 166884 1946	30 M	1 yr.	Hemoptysis, cough, pneumonia; growing hilar mass. Repeated biopsies "benign"	Right main bronchus	Intrabronchial mass 1 × 2 cm. infiltrating wall; largest portion 10 cm. just below bifurcation	None	Leiomyosarcoma bronchus	Died at operation of hemorrhage and asphyxia

AUTHOR YEAR	AGE AND SEX	DURATION OF SYMPTOMS	CLINICAL SYMPTOMS	LOCATION OF TUMOR	GROSS RELATIONSHIP	METASTASES	MICROSCOPIC DESCRIPTION	DEATH OR DURATION
Curry and Fuchs[43] 1950	13 F	6 wk.	Cough, left chest pain, wheezing; 1 wk. before admission, coughed up lump of tissue	Left main bronchus	Gray-red mass, 1.5 × 1 cm., smooth gray surface	None	Fibrosarcoma; numerous mitoses; atypical fibroblasts	4 yr., no recurrence
Carswell and Kraeft[42] AFIP 239010 1950	29	18 mo.	Left chest pain, dyspnea, cough, small hemoptyses, chills, fever	Left main bronchus	Round, smooth, shining non-ulcerated, dark red, fairly firm tumor, treated by bronchoscopy and pneumonectomy	None	Squamous-cell metaplasia of bronchus, spindle-shaped fibroblasts arranged in loose nests or with no pattern; large hyperchromatic nuclei. Pneumonectomy specimen: small portion of sarcoma remaining after bronchoscopy	7½ mo. P.O. normal bronchial mucosae
Black[40] 1950	46 F	9 mo.	Cough, fever, weight loss, hemoptysis	Basal divisions left lower lobe bronchus	Polypoid, 1 × 5 cm. mass, completely occluding bronchus and extending into parenchyma at hilum	None demonstrated at operation	Fibrosarcoma moderately undifferentiated with pleomorphism and mitoses. Fine reticulin fibers around individual cells	Died of massive intrathoracic recurrence 6 yr., 5 mo. P. O.

As shown in Table II, one patient remained well four years after the spontaneous expectoration of a "rapidly growing fibrosarcoma." In another, the lungs removed at autopsy showed only fibrosis at the biopsy site.[48] On the other hand, massive intrathoracic recurrence of another tumor of corresponding histologic appearance resulted in the death of the patient six and one-half years after surgical removal.

Other interesting features of this tumor may be discovered by comparing data from Tables I and II.

From the figures in Table III it is readily seen that the endobronchial sarcomas, in contrast to those of parenchymal origin, occur more rarely and in younger individuals, are discovered earlier in their development, and in behavior are relatively more benign. It may be assumed that any benign or malignant tumor of endobronchial location may easily attract clinical attention; also that parenchymal tumors being "silent" have a much longer preoperative course. However, neither do these factors nor the anatomic location entirely explain the remarkable therapeutic results in patients with "fibrosarcoma" of the bronchus, for similar results are not obtained even when small bronchiogenic carcinomas happen to be removed correspondingly early.

TABLE III. COMPARATIVE DATA FROM CASE REPORTS OF PULMONARY SARCOMA AND ENDOBRONCHIAL SARCOMA

	ENDOBRONCHIAL SARCOMA	PULMONARY SARCOMA	
Number of Cases	8	19	
Sex			
Male	4	14	
Female	4	5	
Duration			
Range	6 weeks-18 months	None-7 years	
Average	7 months	17 months (14 cases)	
Age			
Range	13-46 years	18-65 years	
Average	28 years	46 years	
Location			
Main stem	7		
Upper	0	4	16 cases specified
Middle	0	2	
Lower	1	8	
Combined lobes	0	2	
Right	4	11	
Left	4	8	
Behavior			
Extrathoracic spread	0	4	
Intrathoracic extension or recurrence	1 (after 6 yrs.)	3	

Whether the data in Table III are the accident of a small series, the result of misinterpretation of the histologic criteria for malignancy, or a true indication of differences in behavior will not be clear until there are available larger series with more extensive periods of postoperative observation. Until then the trends revealed by these figures justify, at least, clinical separation of the endobronchial sarcomas from intrapulmonary spindle-cell sarcoma.

Rare Types of Sarcoma of the Lung.—There are few reported cases of primary bronchopulmonary sarcoma other than those formed by spindle cells

of varying degrees of differentiation. Examples of "myxoliposarcoma," "chondromyxosarcoma," "myosarcoma," and "giant cell sarcoma" are recorded.[49-53] However, in these the distribution of the tumor was so widespread that any possible pulmonary origin was obscured, or the tumors were so poorly differentiated that the histogenesis was uncertain.

Malignant tumors composed of cartilaginous elements are reported with more frequency, but they also constitute a poorly defined group.[54] Only in one of the recorded cases was a primary origin demonstrated.[55] I believe the theory could be advanced that the chondroblastic tissue of the bronchus is the source of the tumor rather than a teratoma or hamartoma, as might be suspected from the example reported by Krienitz.[56] In 3,000 primary tumors of the chest filed at the Armed Forces Institute of Pathology, there are no examples of chondrosarcoma in which the tumor is clearly of pulmonary origin.

Similar hesitancy is experienced in the acceptance of reported cases of primary rhabdomyosarcoma, an exceedingly rare but important example of pulmonary sarcoma. Rhabdomyosarcoma metastatic to the lung may simulate a primary growth both clinically and anatomically, thus obscuring the true origin of the tumor in a distant site or in such neighboring tissues as the heart, mediastinum, diaphragm, or upper esophagus.[57-59] On record is a rhabdomyosarcoma arising in the pulmonary artery.[60]

In its usual form, rhabodomyosarcoma of the lung shares with chondrosarcoma a disposition toward vascular spread, a possible component of several histologic cell types, and a tenuous relationship to teratomas or compound tumors in its derivation.[61]

When primary development of rhabdomyosarcoma does occur, it may be explained as a result of misdirection of the mesenchymal cells investing the bronchial buds as illutrated by Zipkin's[62] case in which the bronchial walls contained striated muscle. Dr. Edith L. Potter, in a personal communication, described a loose network of striated muscle fibers in the pulmonary interstitium of a stillborn infant.

These unusual demonstrations of the potentiality of normal cells under abnormal conditions are correlated with the fact that there is only one recorded example of pulmonary rhabdomyosarcoma of unitary cell type.[63] The Chest Tumor Registry also contains one example of well-differentiated rhabdomyosarcoma in which the certainty of a primary pulmonary origin was weakened by an apparent collision in one area with an epidermoid carcinoma of the esophagus.

This tumor is of greater importance than its extreme rarity indicates because it is often considered in the differential histologic diagnosis of certain types of bronchiogenic carcinoma.[64, 65]

DIFFERENTIAL HISTOLOGIC DIAGNOSIS

The imitators of bronchopulmonary sarcoma that are most frequently confused with the true primary tumors are "oat-cell" carcinoma,[66] postinflammatory repair,[67, 68] and pleomorphic-cell carcinoma of the bronchus.[65, 66]

When a peripheral sarcoma encroaches upon a bronchus, it may in the biopsy specimen resemble undifferentiated cell carcinoma of the bronchus ("oat cell" or "reserve-cell" carcinoma) (Fig. 2). The diagnosis is of great importance since the epithelial tumor, according to statistics from the Armed Forces Institute of Pathology, is 500 times as common and is more rapidly fatal to the patient. The frequent diagnosis of sarcoma of the lung in the medical literature may be the result of confusion of these two tumors. Reticulum stains are especially useful in differentiation, since the reserve cell carcinoma was shown in the course of this study to produce no stainable reticulum and each sarcoma, stained at the same time, displayed small or large amounts of this material (Fig. 4).

In other instances inflammatory reactions in the bronchus or pulmonary parenchyma may stimulate such proliferation and repair as to produce a tumor resembling true neoplasia. Brown and Johnson[67] have recently described this process in the pleura resulting from poorly resolved, localized exudate. Somewhat similar examples have been previously reported in the lung,[24, 69-73] and four cases have been more recently described.[68] These, like the peripheral sarcomas, may be well-circumscribed, firm, white, and slow growing. However, they are usually smaller than sarcomas, and the admixtures of inflammatory cells, together with bits of bronchiolar epithelium which may be attracted to the mass, serve to distinguish the puzzling fibroblast as reactive rather than neoplastic. Differentiation may be impossible when portions of the "inflammatory tumor" are composed solely of fibroblasts, colonizing in whorls and duplicating a fibrosarcoma.

The endobronchial sarcomas are among the most difficult to diagnose. Not only may the neoplastic process be masked by secondary inflammation but when biopsy of a small polypoid tumor does reveal a sarcomatous pattern, the pathologist may not be certain whether the tumor is a true sarcoma or an epidermoid carcinoma showing spindle changes (Fig. 12). Ackerman[74] recently described four cases of the polypoid tumor designated "carcinosarcoma" by him and "sarcomatoid carcinoma" by others.[75] This polypoid compound tumor is slow growing, of relatively low malignancy, and its behavior is more like that of true endobronchial sarcoma than like that of orthodox examples of bronchiogenic carcinoma (Fig. 12). Although differentiation may be of only academic importance, it can be made in some instances by multiple sections showing clearly squamous epithelium in transition to sarcomatous structures (Fig. 13).

Although reported examples of rhabdomyosarcoma are extremely rare, their importance lies in the fact that they may be confused with pleomorphic cell carcinoma.[65, 66] In this type of bronchiogenic carcinoma the neoplastic cells become dissociated and bizarre imitating the large "plasmodial" giant cells described as a variant of rhabdomyosarcoma.[76, 77] The differentiation of the two tumors is of both therapeutic and prognostic significance, for a study of nine examples of pleomorphic cell carcinoma reveals that this tumor tends to achieve a large, bulky growth which, by virtue of its relentless local extension or distant metastasis, kills the patient more swiftly. Search for cross striations

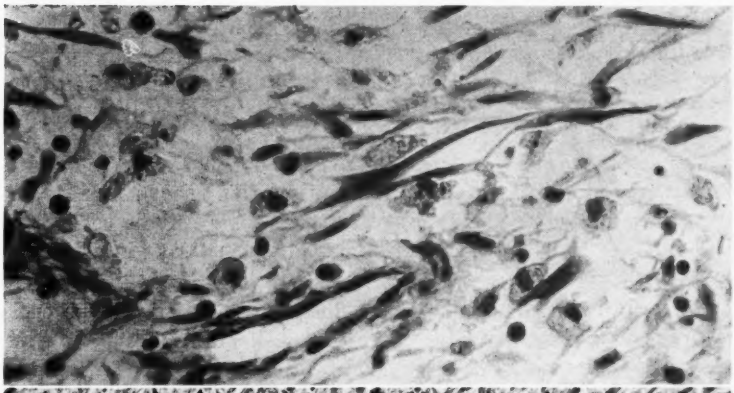

Fig. 11.

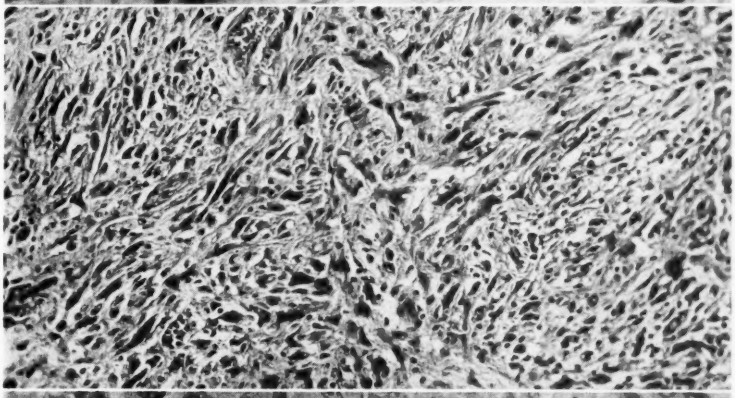

Fig. 12.

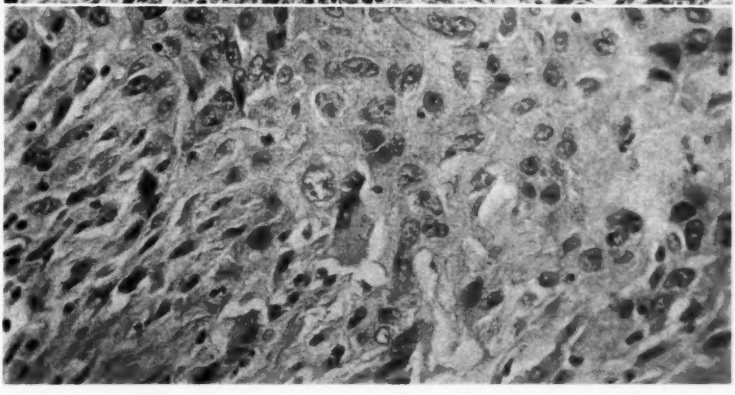

Fig. 13.

Fig. 11.—Endobronchial sarcoma: biopsy specimen from intrabronchial mass, showing fibroblasts, macrophages, and inflammatory cells. Hematoxylin and eosin, ×450. AFIP Acc. 239010.

Fig. 12.—Sarcomatoid epidermoid carcinoma of bronchus. Hematoxylin and eosin, ×125. AFIP Acc. 260561.

Fig. 13.—Sarcomatoid epidermoid carcinoma of bronchus. The sarcomatous appearance of large portions of the tumor is confusing until serial sections show transitions with areas of squamous epithelium (see Fig. 12). Hematoxylin and eosin, ×275. AFIP Acc. 260561.

or for fibers impregnated with silver may aid in distinguishing the two tumors. If these features are absent, multiple sections of these bizarre giant cell tumors will usually reveal mucus production, abortive gland formation, or syncytial patterns, which aid in the accurate diagnosis of the tumor as "carcinoma."

SUMMARY AND CONCLUSIONS

1. The cases reported here are acceptable as pulmonary spindle-cell sarcoma on the basis of clinical and morphologic evidence. This tumor is most frequently a well-circumscribed neoplasm, composed of immature spindle cells; it occurs in middle life, is of relatively long duration, and metastasizes in approximately one-third of the cases.

2. Sarcoma in the proximal bronchi differs from sarcoma of parenchymal origin in that it occurs even more infrequently, affects chiefly young adults, is relatively less malignant in behavior, and is usually shown to be composed of mature fibroblasts.

3. Other types of mesodermal sarcomas, such as liposarcoma, rhabdomyosarcoma and chondrosarcoma are so very rare that only isolated examples have been reported.

4. The accurate morphologic diagnosis of bronchopulmonary sarcoma is complicated by its more frequent imitators. These are: (a) undifferentiated cell bronchiogenic carcinoma, which may be distinguished from poorly differentiated pulmonary sarcoma by reticulum stains; (b) postinflammatory fibroblastic repair simulating neoplasia and resembling well-differentiated pulmonary sarcoma; (c) polypoid epidermoid carcinoma with sarcomatoid differentiation, which in behavior and in structure may imitate endobronchial sarcoma; and (d) pleomorphic cell carcinoma of the bronchus, which is often diagnosed myosarcoma or rhabdomyosarcoma.

REFERENCES

1. Ball, H.: Primary Pulmonary Sarcoma; A Review With Report of an Additional Case, Am. J. Cancer **15**: 2319-2330, 1931.
2. Amorim, A.: Rabdomiosarcoma do pulmão, Rev. brasil. de cir. **11**: 263-280, 1942.
3. Cote, G. L.: Un cas de sarcome primitif du poumon, Laval méd. **10**: 487-499, 1945.
4. Fedoroff, S. P.: Ein operierter Fall von Lungensarkom, Zentralbl. f. Chir. **57**: 707-712, 1930.
5. Lilienthal, H.: Pneumonectomy for Sarcoma of the Lung in a Tuberculous Patient, J. THORACIC SURG. **2**: 600-615, 1933.
6. Bugoslavaskaya, T. V.: Case of Primary Sarcoma of Lung, Vrach. delo. **25**: 463-466, 1945.
7. Carrara, N.: Il sarcoma primitivo (del polmone nell'infanzia), Pediatria **43**: 823-841, 1935.
8. Catron, L. B. S.: Leiomyosarcoma of the Pleura, Report of Case, Arch. Path. **11**: 847-853, 1931.
9. Steward, W. D.: Sarcoma of the Lung. Am. Rev. Tuberc. **40**: 224-228, 1939.
10. Tourreilles, J. F., Pagés, J. M., and Piernes, A.: A propósito de un caso de fibrosarcomatosis, Semana méd. **2**: 1527-1534, 1935.
11. Acuña, M., Winocur, P., and Orosco, G. P.: Sarcoma primitivo de pulmón en una niña de ocho años, Semana méd. **2**: 474-481, 1930.
12. Carrière, G.: Histoire clinique d'un cas d'ictère chronique par rétention par compression des voies biliaires dues à des métastases ganglionnaires périhilaires d'un sarcome primitif du poumon, Echo méd. du Nord **6**: 97-118, 1936.
13. Fabre, J., Bouissou, H., and Pailloncy, B.: Les réticulosarcomes ganglionnaires (a propos de 6 cas), Toulouse méd. **51**: 167-182, 1950.
14. Koukel, A. S.: A Case of Primary Sarcoma of the Lung, Vestnik khir. **55**: 766-769, 1938.

15. Lang, H. B., and Zuerhut, H.: Retothelsarkomatose bei einen 7 Jahre alten Kind, Oesterr. Ztschr. f. Kinderh. **4**: pt. 3-4, pp. 225-236, 1950.
16. Mercader, N.: Consideraciones clínicas sobre un caso de sarcoma de pulmón, Rev. Asoc. méd. argent. **49**: 366-372, 1935.
17. Roberts, O. W.: A Case of Pulmonary Sarcoma, Lancet **1**: 917, 1931.
18. Rosenblum, F., and Gasul, B.: A Case of Primary Sarcoma of the Lung in an Infant Twenty-Nine Months of Age, Arch. Pediat. **48**: 63-65, 1931.
19. Herzmann, K.: Ueber einen Fall von solitärem Spindelzellsarkom der Lunge bei einem 11jährigen Mädchen, Ztschr. f. Kinderh. **59**: 236-238, 1937.
20. Lazer, M.: Zur Klinik und Differentialdiagnose des primären Lungensarkoms, Tuberkulose **13**: 79-83, 1933.
21. Lutembacher, R.: Sarcome du poumon propagé à la veine cave supérieure, Arch. d. mal. du coeur **27**: 94-97, 1934.
22. Edwards, A. T.: The Surgical Treatment of Intrathoracic New Growths, Brith. M. J. **1**: 827-830, 1932.
23. Jackson, C.: Malignant Growths of the Lung; Bronchogenic Diagnosis, Arch. Otolaryng. **12**: 747-752, 1930.
24. Thornton, T. F., Adams, E. E., and Bloch, R. G.: Solitary Circumscribed Tumors of the Lung, Surg., Gynec. & Obst. **78**: 364-370, 1944.
25. Kobylínski, A.: Ueber primäre Sarkome der Lunge, Dissertation, Greifswald, 1904, H. Adler. (Quoted from Adler.)
26. Boschowsky, W.: Ueber primäres Lungensarkom, Frankfurt. Ztschr. f. Path. **9**: 239-257, 1912.
27. Stevens, A. A.: Malignant Disease of the Lung with Special Reference to Sarcoma, Am. J. M. Sc. **144**: 193-202, 1912.
28. Pritchard, J. S.: An Unusual Case of Pulmonary Neoplasm, Am. J. Roentgenol. **8**: 555-559, 1921.
29. Campatelli, A.: Contributo allo studio dei sarcomi primitivi del polmone e loro trattamento, Tumori **12**: 144-160, 1926.
30. Edwards, A. T.: Account of Seven Operable Cases, Intrathoracic New Growths, Brit. J. Surg. **14**: 607-628, 1927.
31. Scheidegger, S.: Eine seltene Lungengeschwulst, Ztschr. f. Krebsforsch. **35**: 172-177, 1932.
32. Mysh, V. E.: Operative Description of Cases of Primary Sarcoma of the Lung, Soviet Klinika **19**: 433-440, 1933.
33. Lyssunkin, I. I.: Zur Frage der primären Bindegewebsgeschwülste der Lungen (Entwicklung eines xanthomatösen Fibrosarkoms aus einem Fibrom der Lunge), Beitr. z. path. Anat. u. z. allg. Path. **94**: 491-500, 1934.
34. Johns, E. P., and Sharpe, W. C.: Primary Pulmonary Sarcoma; Case Report, Am. J. Cancer **23**: 45-51, 1935.
35. Massachusetts General Hospital: Cabot Case #22441. Fibrosarcoma of the Right Lung with Metastases, New England J. Med. **215**: 837-839, 1936.
36. Stohr, R., and Sachs, W.: Zur Klinik und Pathologie des primären Lungensarkoms, Deutsche Ztschr. f. Chir. **249**: 481-491, 1938.
37. Massachusetts General Hospital: Cabot Case #24472. Fibrosarcoma of the Lung, Probably Primary, New England J. Med. **219**: 854-856, 1938.
38. Ellis, R. C.: Primary Sarcoma of the Lung With Brain Metastasis, J. Kansas M. Soc. **40**: 243-245, 1939.
39. Massachusetts General Hospital: Case #31271. Fibrosarcoma of the Lung, New England J. Med. **233**: 18-22, 1945.
40. Black, H.: Fibrosarcoma of the Bronchus, J. THORACIC SURG. **19**: 123-134, 1950.
41. Randall, W. S., and Blades, B.: Primary Bronchogenic Leiomyosarcoma, Arch. Path. **42**: 543-548, 1946.
42. Carswell, J., Jr., and Kraeft, N. H.: Fibrosarcoma of the Bronchus, J. THORACIC SURG. **19**: 117-122, 1950.
43. Curry, J. J., and Fuchs, J. E.: Expectoration of the Fibrosarcoma, Patient Well Four Years Later, J. THORACIC SURG. **19**: 135-137, 1950.
44. Baum, O. S., Richards, J. H., and Ryan, M. D.: A Case of Atelectasis of the Right Lower and Middle Lobes with Bronchoscopy Demonstrating Spindle Cell Sarcoma of the Right Main Bronchus, Ann. Int. Med. **12**: 699-708, 1938.
45. Massachusetts General Hospital: Cabot Case #24202. Presentation of Case, New England J. Med. **218**: 843-845, 1938.
46. Pollack, B. S., Cohen, S., Borrone, M. G., and Gnassi, A. M.: Primary Sarcoma of the Bronchus, Case Report, Am. J. Roentgenol. **41**: 909-914, 1939.
47. Lewis, I.: Sarcoma of Bronchus, Proc. Roy. Soc. Med. **40**: 119-120, 1946.
48. Baum, O. S., Silverman, G., Rosh, R., and Riley, R. L.: A Case of Spindle Cell Sarcoma of the Bronchus (Supplementary Report), Ann. Int. Med. **17**: 750-754, 1942.

49. Latienda, R. I., and Itoiz, O. A.: Mixoliposarcoma del pulmon, Arch. Soc. argent. de anat. norm. y pat. **8:** 563-570, 1946.
50. Lemon, W. S.: Rare Intrathoracic Tumors, M. Clin. North America **15:** 17-46, 1931.
51. Ausbüttel, F.: Primäres Lungenvensarkom, Frankfurt. Ztschr. f. Path. **53:** 303-312, 1939.
52. Jessop, W. J. E.: Sarcoma of the Lung, Irish J. M. Sc., pp. 171-172, April, 1928.
53. Russolillo, M.: Sopra un caso di condromixosarcoma del pulmone (studio clinico ed anatomo-istologico), Riv. di chir. **2:** 128-141, 1936.
54. Lowell, L. M., and Tuhy, J. E.: Primary Chondrosarcoma of the Lung, J. THORACIC SURG. **18:** 476-503, 1949.
55. Greenspan, E. B.: Primary Osteoid Chondrosarcoma of the Lung: Report of a Case, Am. J. Cancer **18:** 603-609, 1933.
56. Krienitz, C. D. W.: Ein Fall von Adenoma der Lunge, Inaugural Dissertation, Friedrichs-Universität, Halle, Willenberg, C. A. Kaemmerer, 1903.
57. Danciger, J. A., and Warren, J. W.: Rhabdomyosarcoma, Case Report, J. Pediat. **19:** 223-228, 1941.
58. Martin, J. F., and Levrat, M.: Rhabdomyosarcome du rein généralisé au poumon et à la surrénale, Lyon méd. **153:** 577-581, 1934.
59. Bosse, M. D.: Rhabdomyosarcomatous Pulmonary Metastases from a Teratoma of the Testis, Am. J. Cancer **39:** 343-346, 1940.
60. Durgin, B., and Ingleby, H.: Primary Sarcoma of the Pulmonary Artery, Clinics, **5:** 182-189, 1946.
61. Helbing, C.: Ueber ein Rhabdomyom an der Stelle der linken Lunge, Centralbl. f. allg. Path. u. path. Anat. **9:** 433-439, 1898.
62. Zipkin, R.: Ueber ein Adeno-Rhabdomyom der linken Lunge und Hypoplasie der rechten bei einer totgeborenen Frucht, Virchows Arch. f. path. Anat. **187:** 244-264, 1907.
63. McDonald, S., Jr., and Heather, J. C.: Neoplastic Invasion of the Pulmonary Veins and Left Auricle, J. Path. & Bact. **48:** 533-543, 1939.
64. Foot, N. C.: Pathology in Surgery, Philadelphia, 1945, J. B. Lippincott Company.
65. Smetana, H. F., Iverson, L., and Swann L. L.: Bronchogenic Carcinoma, Mil. Surgeon **111:** 335-350, 1952.
66. Saphir, O., and Vass, A.: Carcinosarcoma, Am. J. Cancer **33:** 331-361, 1938.
67. Brown, W. J., and Johnson, L. C.: Postinflammatory "Tumors" of the Pleura. Three Cases of Pleural Fibroma of the Interlobar Fissure, Mil. Surgeon **109:** 415-424, 1951.
68. Umiker, W. O., and Iverson, L.: Inflammatory Tumors of Lung. (In preparation.)
69. Brewer, L. A., III, Jones, W. M. G., and Dolley F. S.: Nonmalignant Intrathoracic Lesions Simulating Bronchiogenic Carcinoma. Report of Thirty Operated Cases, J. THORACIC SURG. **17:** 439-463, 1948.
70. Brunn, H.: Two Interesting Benign Tumors of Contradictory Histopathology, J. THORACIC SURG. **9:** 119-131, 1940.
71. Ford, W. B., Thompson, C. W., and Blades, B.: Xanthoma of the Lung, Postgrad. Med. **8:** 48-50, 1950.
72. Freelander, S. O., and Wolpaw, S. E.: Chronic Inflammatory Lesions of the Lung Simulating Bronchiogenic Carcinoma, J. THORACIC SURG. **9:** 530-543, 1940.
73. Scott, H. W., Jr., Morrow, A. G., and Torrence, P. B.: Solitary Xanthoma of the Lung, J. THORACIC SURG. **17:** 821-825, 1948.
74. Ackerman, L. V., and Taylor, F. H.: Neurogenous Tumors Within the Thorax; A Clinicopathological Evaluation of Forty-Eight Cases, Cancer **4:** 669-691, 1951.
75. Rock, D. A., and Hall, J. W.: Squamous Cell Carcinoma of the Bronchus and Spindle Cell Sarcoma of the Lung, Proc. New York Path. Soc., pp. 16-19, 1942-1945.
76. Gray, S. H., and Gruenfeld, G. E.: Myoblastoma, Am. J. Cancer **30:** 699-708, 1937.
77. Rakov, A. I.: Malignant Rhabdomyoblastomas of the Skeletal Musculature, Am. J. Cancer **30:** 455-476, 1937.

EXPERIMENTAL PRODUCTION OF PROGRESSIVE PULMONIC STENOSIS

DAVID E. DONALD, B.V.S., M.R.C.V.S.,* AND JOHN W. KIRKLIN, M.D.**
ROCHESTER, MINN.

TO DATE no one has described an ideal method for the gradual occlusion of a large vessel wherein the degree of occlusion present at a given time could be predicted; this lack occurs in spite of the facts that the problem has attracted attention for many years and that a variety of methods have been employed in pursuit of this goal. Reports on the use of metal bands,[1] fascial strips,[2] ligatures of cotton or silk,[3] the excision of part of the vessel wall,[4] and the application of dicetyl phosphate[5] or of Cellophane film[6] are found in the literature; various degrees of success have been claimed for the respective methods.

In our laboratory, the method of wrapping of large veins with bands of Cellophane has been used routinely to produce gradual obliteration of the lumina of vessels; others[7] have reported progressive narrowing of the thoracic or abdominal aorta in dogs by use of similar methods. Results of the application of such bands to the main pulmonary artery or around the outflow tract of the right ventricle in dogs form the subject of this report.

METHODS

In the first group of animals, the Cellophane was applied around the main pulmonary artery in its middle third. An incision was made in the left side of the thorax between the fourth and fifth ribs. The pulmonary artery was thus exposed and was completely freed from its attachment to the aorta by sharp dissection close to the pulmonary arterial wall. One or two laminated Cellophane bands, 6.5 mm. in width, were placed completely around the vessel. In the first two dogs in this group, the band simply was fitted snugly around the artery, but in subsequent operations it was tightened so as to obtain an initial reduction of the lumen estimated to be 50 per cent of the original cross-sectional area. Following this procedure and after a period of observation of the heart's action, the pericardium was closed and the thoracic wall restored in layers.

The same methods of approach and closure were used in the animals of the second series, but in this group of dogs a tunnel was formed by dissection beneath the pericardial fat pads that occupy the region of the fibromuscular junction of the pulmonary artery and right ventricle. Two or three Cellophane bands were passed through this tunnel so as to encircle completely the pulmonary artery; again, an estimated 50 per cent reduction of the lumen was sought. By this method the bands were secured in position at the ring of the pulmonary valve and were prevented from riding up the artery.

In the final group of animals, an attempt was made to produce subpulmonic stenosis. A midsternal incision was made in the thorax and the heart was cradled by pericardial stay sutures. A finger was inserted into the cavity of the right ventricle through a stab wound made approximately 6 cm. below the ring of the pulmonary valve. With this finger utilized as a means of control, a long, slightly curved needle was driven through the interventricular septum 1.5 to 2 cm. from the pulmonary valve and made to emerge beneath

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*Fellow in Physiology, Mayo Foundation.
**Division of Surgery, Mayo Clinic.

the right coronary artery 1 to 1.5 cm. from the ring of the valve. A Cellophane band, 6.5 mm. in width, was attached to the needle and pulled through the interventricular septum. The band was then threaded across the lateral aspect of the wall of the right ventricle in a series of short bites until the original point of entrance was reached. The band was tied under some degree of tension and the knot secured with a cotton ligature. The pericardium was closed with interrupted sutures and the thoracic wall restored.

Preoperative roentgenograms and electrocardiograms were obtained, and these studies were continued in the postoperative period. Catheterization of the right side of the heart was performed at intervals throughout the time of observation; the pressures were recorded by means of a saline manometer. In some animals, the final readings were obtained by means of a strain-gauge galvanometer system similar to that described by Wood.[8]

RESULTS

Placing the Cellophane bands at operation was not without hazard; instances were encountered in which a tear was occasioned in the thin-walled pulmonary artery. Experience demonstrated that freeing of the vessel was accomplished best by dissection with sharp scissors in the plane of tissue between the arterial wall and its overlying fibrofatty tissue, aided by blunt dissection during which the points of a right-angled gall bladder clamp were worked against the tip of the finger. Also, it was not easy to judge the correct amount of initial constriction; overzealous immediate tightening of the band resulted in death from excessive obstruction to the outflow of the right ventricle.

Instances of what might be interpreted as pulmonary vasoconstriction were encountered in two dogs. During dissection, the pulmonary artery suddenly decreased greatly in size; this was followed quickly by ballooning of the right ventricle and a noticeable slowing and weakening of cardiac action. This state was maintained for three or four minutes, when an equally sudden return of the pulmonary artery to its previous size occurred, accompanied by rapid disappearance of the right ventricular dilatation and resumption of normal cardiac action. One of these two dogs was found dead the morning after operation; findings at necropsy were indicative of acute obstruction to outflow from the right ventricle.

It was noted also that only one or two bands should be placed. If more than this number were applied, they combined to form a hard, unyielding ring that ultimately eroded through the wall of the pulmonary artery.

PULMONIC STENOSIS

Studies of Pressure.—The dogs in the first group were catheterized twice, once within a month of operation and again just prior to death. The results are given in Table I. Right ventricular hypertension was demonstrated in two of four surviving dogs. These two animals were among those in which initial reduction in the lumen of the pulmonary artery had been obtained. Pressures in the pulmonary artery distal to the band, in the right auricle, and in the inferior vena cava were within normal limits.

The animals in the second group were catheterized more frequently; the results are listed in Table II. Two dogs showed slight increase of right ventricular pressure early in the postoperative period, but a progressive increase

TABLE I. MEAN BLOOD PRESSURES IN 4 DOGS AFTER SIMPLE PLACEMENT OF CELLOPHANE BANDS AROUND PULMONARY ARTERY

	PRESSURES, MM. OF MERCURY				REDUCTION	
DAYS P.O.	INFERIOR VENA CAVA	RIGHT AURICLE	PULMONARY ARTERY	RIGHT VENTRICLE	IN LUMEN, PER CENT	HYPERTROPHY
240	3	4	20	39	76	Present
210	4.4	7	*	46	81	Extensive
212	2.2	0.6	*	13	40	Present
224	3	0.9	16	13	39	Absent

*Unable to pass catheter into pulmonary artery.

was revealed during later studies. Right ventricular hypertension was present finally in three of the four dogs in this group; again, pressures in the pulmonary artery and right auricle remained within normal limits. No evidence was noted of elevation of the end-diastolic pressure in the right ventricle. One of the animals which exhibited increased pressure in the right ventricle early in the postoperative period was found dead on the nineteenth postoperative day; the wall of the pulmonary artery had been eroded by the constricting Cellophane band.

TABLE II. MEAN BLOOD PRESSURES (EXCEPT WHERE INDICATED) IN 4 DOGS AFTER ANCHORING CELLOPHANE BANDS AROUND PULMONARY ARTERY

		PRESSURES, MM. OF MERCURY				REDUCTION	
DOGS	DAYS P.O.	INFERIOR VENA CAVA	RIGHT AURICLE	PULMONARY ARTERY	RIGHT VENTRICLE	IN LUMEN, PER CENT	HYPERTROPHY
1	16	9	6	17	35		
	57	7	6	19	34		
	117	–	4/–1	18/10	95/1	77	Present
2	22	4	1	13	14		
	64	2	0.7	11	15		
	93	6/2	5/1	24/8	60/1	63	Slight
3	12	14.5	14.5	34	26	Pulmonary artery eroded by band	Present
4	13	4	1.5	18	14		
	37	2.6	1	21	20		
	97	–	–3/–5	30/12	29/–3	64	None

Electrocardiograms.—No changes were observed in postoperative records until the third day. At this time, slight elevation of the S-T segment was noted in Leads I, II, III, and aV_F and depression of the S-T segment of equal magnitude was present in leads aV_R and aV_L; these changes were accompanied by an increase in the amplitude of the T wave in all leads. These alterations progressed to a maximum on the seventh day, when the shift of the S-T segment was well defined and the size of the T wave was equal to, or exceeded, that of the QRS complex. From this point the modifications regressed until, on the seventeenth postoperative day, the S-T segment was again on or near the base line and the T wave was the same size as that in control records. Beginning at this time, gradual alterations of the QRS complex were noted, the most prominent features being an increase in depth of Q_I, S_{II}, and S_{aVF}, with increase in height of R_{aVR} and decrease of S_{aVR}. These variations progressed to produce final records that were greatly different from control records, a

mainly negative deflection being noted in Leads II and aV_F, while aV_R had become wholly positive. A biphasic deflection of approximately equal negative and positive components was recorded from Leads III and aV_L. In these last records, a positive shift occurred in the S-T segment in Leads II, III, and aV_F, with an accompanying negative displacement in lead aV_R. No significant alteration was noted in the duration of the QRS interval at any time during the period of observation. An example of these changes in configuration of the QRS complex is seen in Fig. 1.

Animals in which right ventricular hypertension did not develop yielded postoperative records similar to those obtained during the preoperative period.

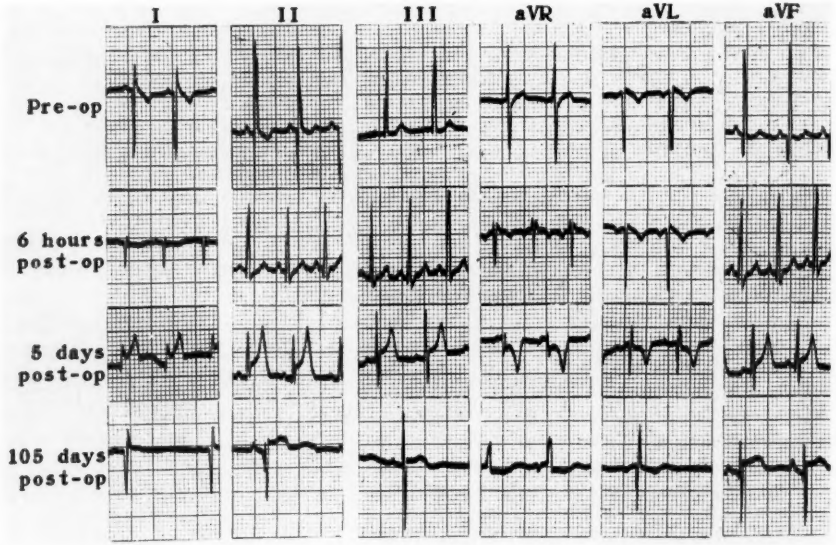

Fig. 1.—Electrocardiographic changes during development of right ventricular hypertension.

Roentgenograms.—In only one dog was any evidence of cardiac enlargement obtained by roentgenologic examination. In this animal, slight enlargement of the cardiac shadow was seen toward the end of the first month; definite evidence of increase in size was present at the beginning of the third month. The records were made with the animal in the anteroposterior position, and the increase in size of the cardiac shadow was noted along the lateral border of the right ventricle. This animal exhibited the greatest right ventricular pressure recorded in this study.

Necropsy and Histologic Findings.—In all dogs the Cellophane bands were buried in a mass of firm, fibrous tissue that was adherent to neighboring structures. Although this connective tissue was applied closely to the Cellophane, it was not adherent to the bands and they could be extracted from

their beds with little difficulty. Apart from being somewhat more brittle than before, the bands were unaltered grossly. In the first series of animals, the bands had moved toward the bifurcation of the pulmonary artery, so that compression of the right and left branches was present at their origin, rather than compression of the main pulmonary trunk. In the second group of animals, the original placement essentially was maintained. In all instances, the cross-sectional area of the lumen of the vessel was reduced at the point of application of the band; measurement of this constriction revealed that only when the lumen was reduced by at least 63 per cent of its original size was right ventricular hypertension present. One exception was noted; one dog, which had no evidence of right ventricular hypertension on catheterization and which had a luminal reduction estimated at 40 per cent, had hypertrophy of the right ventricle at necropsy.

The pulmonary artery beneath the band usually appeared normal grossly, although it was thinner than in immediately adjacent parts. Small ridges of endothelium were present at the point of greatest constriction in two dogs which had hypertension. In addition, an oval aneurysmal area, 1 by 0.5 cm., was found in one of these dogs; this aneurysmal change was situated in the posterior wall of the pulmonary artery approximately 2 or 3 mm. on the right ventricular side of the bands. Prestenotic or poststenotic dilatation of the pulmonary artery was not found in any animal.

In dogs displaying hypertension, the right ventricle was enlarged and overhung the left so that the length from base to apex was equaled by the widest diameter. Measurements of the width of the muscle of the right ventricle demonstrated an increase in the muscle mass of the hearts of hypertensive dogs when compared to hearts of normal dogs or of those in which hypertension did not develop (Fig. 2).

Histologically, compression and atrophy of the pulmonary arterial wall were noted beneath the band, associated with a subendothelial increase in connective-tissue elements, especially on the right ventricular side of the constriction. No alteration was found in the endothelial lining throughout the main pulmonary vessels.

SUBPULMONIC STENOSIS

In no instance was evidence of right ventricular hypertension or hypertrophy found in dogs which had subpulmonic stenosis, nor had the encircling Cellophane band produced any obstruction to outflow from the right ventricle. The bands were found encased in a small rim of dense fibrous tissue, sharply defined from the surrounding muscle; in only one animal was the band found to be broken. One dog exhibited a small outpouching of the wall of the right ventricle between the band and the ring of the pulmonary valve, but catheterization studies revealed no obstruction to outflow from the ventricle.

Microscopic examination of the parts through which the bands had passed revealed that an encircling ring of densely packed, practically avascular,

collagenous fibers had been laid down around the band. This layer of fibrous tissue was distinct from the adjacent cardiac muscle, which exhibited no abnormal features.

COMMENT

Though the series studied was relatively small, points of technique contributing to success or failure were carefully considered in each case. It was

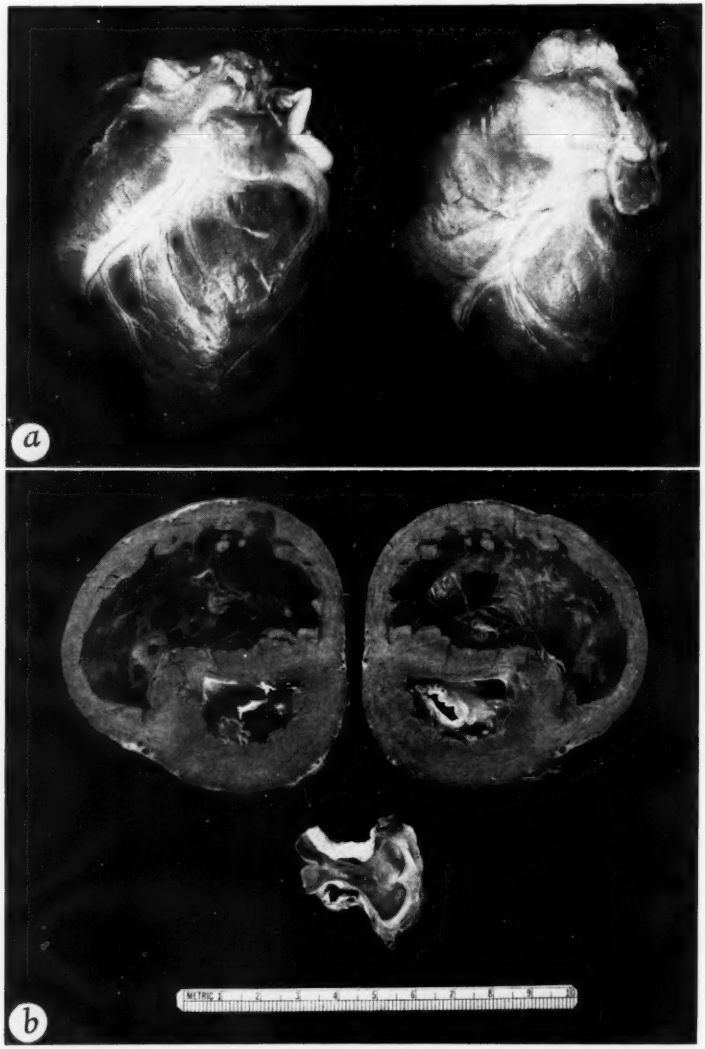

Fig. 2.—*a*, Anterior view of hearts. The specimen on the left was from a dog which did not exhibit right ventricular hypertension; that on the right was from an animal which exhibited a mean right ventricular pressure of 46 mm. of mercury; *b*, cross section of a heart from a dog which had right ventricular pressure of 95/1, expressed in mm. of mercury; note dilatation and hypertrophy of the right ventricle and the degree of stenosis of the pulmonary artery.

considered that the major factor in eventual production of right ventricular hypertension resided in the degree of initial constriction produced at operation; if this was insufficient, the later stricture did not offer enough resistance to outflow from the right ventricle to promote hypertension, while if it was too severe, the right ventricle was unable to maintain circulation against the excessive load. Gibbon and co-workers[9] have shown that the cross-sectional area of the pulmonary artery may be reduced by approximately 60 per cent before hemodynamics are altered. On the basis of this and other work,[10] the figure of 50 per cent was used as the goal of initial constriction in this series, but unfortunately no method was available to insure that this degree of narrowing was obtained in all cases.

That the stenosis was of a progressive character was evidenced by results of the catheterization studies. Records obtained soon after operation were similar to control records; several weeks passed before a substantial increase in right ventricular pressure was noted. It was interesting to find that even in a dog which demonstrated a right ventricular systolic pressure of 100 mm. of mercury no elevation was found in the end-diastolic pressure, while the pressures within the right auricle and the vena cavae remained within normal limits. This situation denotes that the ventricle had fully compensated for the increased resistance to outflow. This compensation was accomplished in all cases without the development of a gross degree of muscular hypertrophy. Similar findings were reported by Holman and Beck,[11] who noted that an increase in flow was a more potent stimulus to cardiac hypertrophy in animals than was resistance to outflow. All dogs showed a decrease in pressure across the region of stricture; the degree of stenosis was paralleled by the amount of right ventricular hypertension.

No immediate electrocardiographic changes of importance were noted after the bands were placed around the pulmonary artery, but later observations revealed two interesting periods of change. During the first period, which lasted from about the third to the seventeenth postoperative day, electrocardiographic findings were compatible with those of right ventricular strain as described by Goldberger.[12] It is possible that the early and vigorous reaction of tissue to the presence of Cellophane produced a temporary stricture of the pulmonary artery that was sufficient to embarrass the right ventricle; then, as the early changes of inflammation regressed, the lumen again widened sufficiently to cause little resistance to outflow from the right ventricle. Unfortunately, no studies of pressure were made during the acute phase of this period. The second phase commenced during or shortly after the terminal stages of the first, and the major changes were noted in Leads II, III, aV_R, and aV_F. These alterations consisted of almost complete reversal in direction of the major QRS component in each lead and were progressive from week to week, finally becoming stabilized between the seventieth and ninetieth days. Comparison between similar leads from these hypertensive dogs and from a 15-month-old greyhound that had congenital pulmonic stenosis and extensive hypertrophy of the right ventricle demonstrated a close resemblance

among the records. The thoracic leads in each instance fulfilled many of the criteria necessary for the electrocardiographic diagnosis of hypertrophy of the right ventricle in man.[13]

Histologically, thinning and atrophy of the pulmonary arterial wall were noted beneath the band, together with an increased amount of subendothelial connective tissue mainly on the central but also on the peripheral side of the region of stenosis. In one dog, a small area of erosion was noted in the arterial wall, presumably caused by turbulent flow created by increased resistance. Otherwise no extensive changes occurred in the pulmonary artery.

SUMMARY

Wrapping of Cellophane bands around the pulmonary artery in dogs resulted in right ventricular hypertension and hypertrophy of the right ventricle. The major factor contributing to a successful end result was thought to be the initial degree of constriction produced at operation.

Results of cardiac catheterization and of electrocardiographic studies were indicative of the progressive nature of the stenosis.

REFERENCES

1. Halsted, W. S.: The Partial Occlusion of Blood-vessels, Especially of the Abdominal Aorta, Bull. Johns Hopkins Hosp. **16:** 346-347, 1905.
2. Pearse, H. E., Jr.: The Impracticability of Using Fascia for the Gradual Occlusion of Large Arteries, Am. J. Surg. **16:** 242-246, 1932.
3. Holman, Emile: Hemicardiac Hypertrophy Due to Increased Peripheral Resistance: A Study of Pulmonic and Aortic Stenosis Experimentally Produced, J. THORACIC SURG. **9:** 262-273, 1940.
4. Hufnagel, C. A., Roe, B. B., and Barger, A. C.: A Technique for Producing Pulmonary Artery Stenosis, Surgery **29:** 77-81, 1951.
5. Berman, J. K., Boyd, F. J., Jr., and Saint, W. K.: The Treatment of Thoracic and Abdominal Aneurysms of Syphilitic and Arteriosclerotic Origin: (Preliminary Report), J. Indiana M. A. **42:** 889-893, 1949.
6. Pearse, H. E.: Experimental Studies on the Gradual Occlusion of Large Arteries, Ann. Surg. **112:** 923-933, 1940.
7. Clatworthy, H. W., Jr., Sako, Yoshio, Chisholm, T. C., Culmer, Charles, and Varco, R. L.: Thoracic Aortic Coarctation: Its Experimental Production in Dogs, With Special Reference to Technical Methods Capable of Inducing Significant Intraluminal Stenosis, Surgery **28:** 245-272, 1950.
8. Wood, E. H.: Special Instrumentation Problems Encountered in Physiological Research Concerning the Heart and Circulation in Man, Science **112:** 707-715, 1950.
9. Gibbon, J. H., Jr., Hopkinson, Mary, and Churchill, E. D.: Changes in the Circulation Produced by Gradual Occlusion of the Pulmonary Artery, J. Clin. Investigation **11:** 543-553, 1932.
10. Fineberg, M. H., and Wiggers, C. J.: Compensation and Failure of Right Ventricle, Am. Heart J. **11:** 255-263, 1936.
11. Holman, Emile, and Beck, C. S.: The Physiologic Response of the Circulatory System to Experimental Alterations. III. The Effect of Aortic and Pulmonic Stenoses, J. Clin. Investigation **3:** 283-298, 1926.
12. Goldberger, Emanuel: Unipolar Lead Electrocardiography: Including Standard Leads, Augmented Unipolar Extremity Leads and Multiple Unipolar Precordial Leads, and a Section on Cardiac Arrhythmias, ed. 2, Philadelphia, 1949, Lea & Febiger.
13. Myers, G. B., Klein, H. A., and Stoffer, B. E.: The Electrocardiographic Diagnosis of Right Ventricular Hypertrophy, Am. Heart J. **35:** 1-40, 1948.

LUNG ABSCESS AND BRONCHIAL CATHETERIZATION

H. METRAS, M.D., AND J. CHARPIN, M.D.
MARSEILLE, FRANCE

I. BRONCHOGRAPHY BY CATHETERIZATION IN LUNG ABSCESSES

WE SHALL deal only with lung abscesses apparently due to no preexisting lesions, such as bronchial carcinoma, neighboring bronchiectasis, hydatid cyst, etc.

It is customary to say[26, 28, 34] that bronchography does not inject the cavity of such abscesses. That is true as far as the classical techniques of bronchial opacification are concerned. On the other hand it is possible to obtain satisfactory opacification of lung abscesses by injecting the contrast medium by means of a catheter into the diseased lobe or segment. We deem such a procedure to be highly profitable since it gives us an accurate anatomic picture of the lesions at the beginning of treatment; adequately localized antibiotic instillations are thereby made possible. At the end of treatment it contributes to the correct appreciation of the final results. Applied to a whole series of lung abscesses, it enabled us to get a clearer picture of their anatomic evolution and in particular to make apparent the remarkable frequency of residual cavities.

We shall successively deal with our equipment, the technique of accurate bronchography in abscesses, and the subsequent results.

EQUIPMENT

The catheters (Fig. 1) that we use* are made of rather stiff rubber. The end of the catheter is made opaque by the addition of lead salts. They allow a deeper and more accurate penetration into the bronchial tree than Thomson's catheter. They come in two diameters: No. 19, the larger of the two, convenient for aspirations and total bronchography with water-soluble medias, and No. 15, the smaller, which is used in most cases.

Different types of catheters may be obtained: *Straight catheter. Curved catheter* whose end makes an angle of approximately 100 degrees with the body. This type of catheter is most often used; in total bronchography it makes it possible to switch rapidly from the right to the left side, and its extremity lodges easily at the entrance of the upper lobe bronchus. It is also used for the middle lobe, and the various segments of the lower lobe (with the exception, however, of the apical segment). *Strongly curved, hook-shaped catheter* convenient for catheterizing the apical segment of the upper lobes. *Catheters with a double bend,* necessary to penetrate the posterior and anterior segments of the upper lobe and often quite useful to enter the bronchus leading to Nelson's lobe. For instance, the same catheter will be used for the posterior segment

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*Designed by H. Metras and manufactured by the firm Gentile in Paris.

of the right upper lobe, for the lingula of the left upper lobe, and for the apical segment of the right lower lobe.

A sensible choice of sound plays a considerable part in the final success. Several attempts are sometimes necessary to hit upon the right type of sound. In some cases, no sound has the curve necessary and it must be modified for the case in question.

Before finishing we would like to point out the ingenious little instrument, devised by Pieri and Casalonga.[67] It is a catheter whose tip can be directed and which can render real service in the catheterization of segments difficult to enter (in particular the apical segment of the superior lobe). Gidlund of Stockholm suggested the use of a similar catheter.

A steel mandrel is used to guide the catheter down to the glottis.

A conical adaptor, because of its cylindrical grooves, allows a satisfactory tightness of the syringe-sound group.

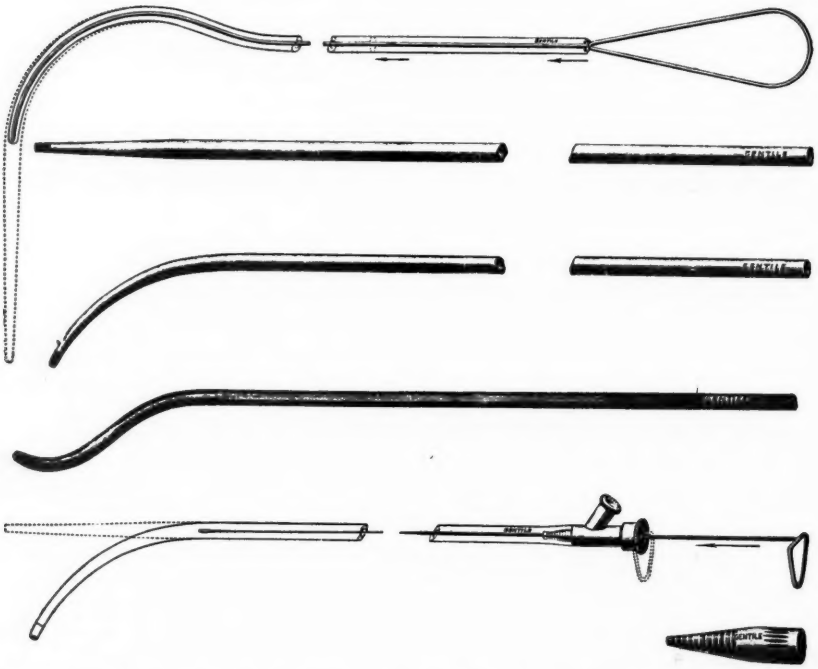


Fig. 1.—Equipment, from top to bottom: the sound on its mandrel, a straight catheter, a curved catheter, a double curved catheter, another type of mandrel, the conical adaptor.

TECHNIQUES

A. Anesthesia.—We use a preanesthesia: 0.20 Gm. gardenal and 0.5 mg. atropine one-half an hour before the examination, then a 1 per cent solution of Pantocaine above the larynx and a 0.5 per cent solution in the bronchial stage.

The use of a Vilbiss atomizer allows a notable saving of drugs in the bucco-pharyngo-laryngeal stage. The total dose of Pantocaine used must not exceed 0.001 Gm. for each kilogram of weight. If this rule is adhered to and if barbiturates are used as preanesthetics, toxic accidents are negligible.

B. Catheterization.—When the anesthesia is complete the patient is made to strip to the waist. The selected catheter is slipped over the guiding mandrel previously lubricated with Lipiodol (we make sure that the catheter moves freely along this mandrel) then with the help of a laryngeal mirror the end of the catheter is placed at the entrance of the larynx. All we have to do then is slip the catheter off the mandrel while holding the latter in position. When the passage of the sound is difficult, as in the cases of catheters with double curves, it is advisable to push the catheter beyond the glottis with the help of the laryngeal mirror; in this instance the catheter should be pushed in by an assistant.

Before leaving the site of anesthesia, we make sure that the catheter is correctly placed in the trachea. (One is relatively sure it is well placed if the catheter can be moved freely; the patient can no longer talk.)

The patient is then requested to close his mouth and hold the catheter in his fingers as he would a cigarette. The remaining procedure is conducted under fluoroscopic control by means of small back and forth and revolving motions; the end of the catheter should be introduced into the lobe or the segment to be catheterized. For this procedure the diaphragmatic opening should be very small so that clear front and lateral view of the end of the catheter may be obtained. Once the catheter is placed, the anesthesia is completed with an injection of 1 c.c. of a 0.5 per cent solution of Pantocaine.

C. Bronchography.—The patient's position is of little importance if a cavity has to be injected since the catheter blocks the return of the Lipiodol, and the examination can be made with the patient standing behind the screen. If a more extensive bronchograph is desired, for example, that of a whole upper lobe, we, of course, must take into account the laws of gravity and operate while the patient is in a lying position.

The most classical contrast medium is a 40 per cent solution of Lipiodol but, in those localized bronchographies which are performed in order to inject abscess cavities, we are much in favor of water-soluble drugs, of the Ioduron B,[11, 14, 33] Umbradyl,[22, 23] or Diodone[8, 24] type, whose advantages we have pointed out.[6] Such drugs make possible repeated opacifications of the cavity without undesirable residual shadows. Moreover, Diodone, extremely irritating in total bronchography is, in this case, injected through the trachea and the larger bronchi, that is, through the most sensitive parts of the bronchial tree, by means of the catheter which makes it perfectly tolerated.

With this technique it is possible to inject, though sometimes after a few failures, nearly all lung abscess cavities. The catheter never penetrates the cavity proper; it blocks up its draining bronchus and the opaque medium injected penetrates, wholly or nearly so, the suppurative lesion.

DATA OBTAINED BY BRONCHOGRAPHY IN LUNG ABSCESSES

Bronchography must be done at the beginning and at the end of treatment of lung abscesses in order to be able to appreciate the quality of the healing.

A. Initial Bronchography.—It presents numerous advantages as Mattei clearly pointed out. It definitely localizes the involved segment. It makes possible an accurate anatomic picture of the suppuration to be treated. In some cases, Lipiodol shows lesions that standard x-ray examinations do not at all disclose; for instance, it outlines one of those fistulas created by parenchymatous suppurations between two segments, or even through an interlobar fissure. Such communications prove particularly frequent between the diverse axillary zones.

For the physicians who treat abscesses with instillations, initial bronchography assures a still more valuable function: the contrast medium shows the antibiotic its way. The practitioner knows that if he uses the same catheter held in the same position, he will safely inject penicillin, Aureomycin, or Chloromycetin into the abscess.

It is often advisable with a patient suffering from acute suppuration to postpone a few days such an examination. As soon as he is hospitalized, we submit such a patient to a systemic antibiotic treatment: penicillin, or even better Iodhydrate of the diethylaminoethylic ester of benzyl penicillin, Aureomycin or Terramycin, according to the bacteriologic data. We immediately perform a bronchoscopy; this examination enables us to spot which segmentary opening exudes pus, to get bacteriologic samples in situ and to cleanse the diseased opening with adrenalin. The radiologic file has in the meantime been established with a good lateral film as the master document. We then proceed to make a first instillation which enables us to select the appropriate catheter, and one or two days later we perform our initial bronchography.

If Lipiodol is applied, we shall use a small dose, 5 c.c. at most, so as not to interfere with later radiologic examinations; but, as we have pointed out, it is better to apply diodone to such cases; the images obtained are a little less spectacular (Figs. 2, 5, 9, and 10) but the advantage of an instantaneous resorption is very great in the case of an affection that will have to be watched daily under x-ray examinations.

B. Final Bronchography.—We deem this step to be of a still greater importance than the initial examination. It is indeed necessary to make another anatomic checkup at the end of the treatment (whatever the technique used). Bronchography often gives more accurate results than tomography, as Case 1 shows.

CASE 1.—A tanner, 48 years old, after one week of fever in January, 1951, expectorated a small quantity of putrid sputum. He was hospitalized on Jan. 15, 1951. He expectorated 150 Gm. of pus daily. X-ray findings included a large cavity in the posterior segment of the right upper lobe. After 38 days of systemic penicillin treatment, one million units daily (no local treatment), there was clinical and radiologic recovery. Tomographies taken on Feb. 27, 1951, showed a small suspicious image within 8 cm. of

the posterior aspect. On March 5, 1951, a diodone bronchography showed a residual cavity in the posterior segment of the right upper lobe (Fig. 2). The patient was discharged under observation.

In the final bronchography, there are fewer inconveniences to the use of Lipiodol and it is often interesting to pull back the catheter into the main bronchus after a local injection of the diseased region, in order to make a complete opacification of the diseased lung so as to verify the soundness of the whole bronchial tree.

Fig. 2 (Case 1).—Abscess of the posterior segment of the right upper lobe treated by systemic penicillin; clinical healing. Bronchography at the end of the treatment showed small residual cavity.

At the end of a treatment, bronchography may reveal a *strictly normal* bronchial tree; on the other hand it often makes apparent *residual lesions:* bronchial dislocations and distortions, small local bronchiectasis, and especially residual cavities at the site of the suppurative lesion. When careful bronchographies are performed on patients clinically "healed" of their lung abscess, residual cavities are frequently discovered. We were impressed by the fact that such cavities are often quite well tolerated, provided they are of small volume and in a location allowing drainage. We have nine patients under observation who have been clinically healed for more than two years in spite of considerable anatomic sequelae revealed at the end of the treatment. Cases 2 and 3 are of such a type.

Case 2.—A 55-year-old market fishmonger, an obese woman, was without any respiratory incident. On April 27, 1949, she presented an acute pneumonitis with an abrupt beginning. She was hospitalized on May 10, 1949, in a serious condition which made any

immediate local intervention inadvisable. Under systemic penicillin treatment the patient's general condition improved but purulent expectoration and radiologic symptoms persisted. The first instillation was made on June 1, 1949. Bronchography performed after the fifth instillation showed a large bilocular cavity in an axillary branch of the anterior segment of the right upper lobe. Eight more instillations of one million units penicillin were given. At the beginning of July, the clinical recovery was complete. The patient left the hospital; she was examined again on Jan. 24, 1951. Clinical recovery persisted, and the standard film was normal.

Fig. 3 (Case 2).—Lipiodol bronchography six weeks after clinical healing, small residual cavity.

In Case 2, we have been unable to establish what became of the residual cavity by means of a later bronchography. On the contrary, the interest in Case 3 lies in the fact that segmentary Lipiodol still revealed a residual cavity in a patient who has been well for over two years.

CASE 3.—L. A. Z., 56 years old, was hospitalized in November, 1948. An abscess in the posterior segment of the right upper lobe showed marked improvement after nine instillations of 400,000 units of penicillin. On Feb. 8, 1949, bronchography revealed a residual cavity and bronchial irregularities. After six more instillations there was clinical healing. The patient was examined again in May, 1951, and there was no respiratory manifestation since the patient's abscess. However, the bronchography performed on May 23, 1951, showed two very small residual cavities in the posterior segment of the right upper lobe (Fig. 4).

Obviously there are less favorable cases where either large or poorly drained cavities precipitate a relapse of the suppuration. Here again, bronchography gives an accurate surgical orientation as Case 4 demonstrates.

CASE 4.—D. U. R., a 45-year-old salesman, had an abscess in the apical segment of the right upper lobe in May, 1950. Healing occurred after local penicillin treatment, yet a huge cavity persisted which was easily injected with diodone (Fig. 5). The location of the cavity as well as the patient's rather poor general condition made resection inadvisable at this time. Relapse occurred in January, 1951; a total diodone bronchography injected no cavities; however, an accurate bronchography performed with Pieri's catheter revealed a large residual cavity. Lobectomy was performed after cleansing instillations (Dr. Reboud). Histologic examination revealed a nonepithelized intraparenchymatous cavity.

CONCLUSIONS

In the treatment of lung abscesses, accurate bronchography, using preferably a water-soluble contrast medium, yields valuable information on the size and the exact location of initial lesions as well as on the amount of anatomic healing.

Fig. 4.

Fig. 5.

Fig. 4 (Case 3).—Abscess of the posterior segment of the right upper lobe healed by instillations. On May 23, 1951, two years and one-half after the clinical healing, bronchography still injects two small residual cavities.

Fig. 5 (Case 4).—Abscess of the apical segment of the right upper lobe, treated by instillations in May, 1950. Diodone bronchography on July 31, 1950, huge residual cavity.

II. TREATMENT OF LUNG ABSCESS BY MEANS OF CATHETER INSTILLATIONS

Since 1945 we have used the catheter just described for therapeutic purposes. This method proved most effective in the treatment of lung abscess. The technique used was the same as the one described previously; the same catheter, and the same position that made a satisfactory opacification of the cavity possible at the time of the initial bronchography were used. When the catheter was in the correct position, the patient standing behind the fluoroscopic screen, anesthesia completed by injecting 1 c.c. Pantocaine, and the patient placed in such a position as to allow the injected fluid to remain as long as possible in the suppurative focus, the therapeutic substance was then slowly injected.

For a long while, the antibiotic used was penicillin alone (one million units in a 3 c.c. saline solution). Actually penicillin remains active in suppurations

caused by anaerobes and often in those caused by gram plus bacteria. If a study of the organisms reveals a poor sensitivity to penicillin, sulfonamids shall be used (1 gr.) or Aureomycin (0.10 Gm. plus sodium glycinate) or Chloromycetin (0.25 Gm.), but this latter is much more irritating for the bronchial tree. Thus we very often use a mixture of one million units of penicillin plus 0.10 Gm. Aureomycin plus 1 Gm. sulfadiazine for the same instillation.

Without neglecting the useful action of systemic antibiotics and of postural drainage, instillations are made every day or every other day. As a rule the patient becomes accustomed to them easily; as soon as the second or third instillation we have performed them without any preanesthesia. The patient's temperature very frequently shows a slight rise in the evening of the day when the instillation is made, but this is not alarming.

When the instillations prove effective, they do so promptly and their action can be felt in the decline of temperature and less abundant sputum. The total number of instillations can vary between six and about twenty; however, they should be discontinued if a marked improvement has not appeared after six or eight instillations. Most of our hospitalized patients have been submitted, prior to their admission, to treatment by several systemic antibiotics or aerosols. Frequently, we have found that the very same antibiotic that has hitherto proved ineffective, becomes effective when injected right into the suppurating focus. It is quite likely that the sort of "chimney-sweeping" effected by the catheter as it penetrates into the draining bronchus has an importance of its own apart from the antibiotic action.

Since initial bronchography enables us to make sure that the substance is correctly injected into the abscess, we think our method as accurate and less dangerous than the transparietal injections favored by many authors.[15, 19, 22, 26] The use of a rubber catheter (placed with the patient behind the fluoroscopic screen) also seems to be preferable to the use of a bronchoscope,[17, 18, 28] since repeated bronchoscopies have a more traumatizing effect than instillations and because an injection performed through a bronchoscope, no matter how skillful the physician, is much less accurate than a catheter injection.

We have eliminated from our statistics all suppurations secondary to a pre-existing bronchopulmonary lesion: hydatid cyst, bronchial carcinoma, bronchiectasis, etc. We are concerned only with apparently primary abscess. We have also had to eliminate about fifteen cases in which our data were too incomplete and cases in which we lost contact with the patients. The remaining fifty-one cases between 1946 and 1952 form the basis of this study. All these patients were examined again in 1951 or 1952 by one of us and many have been submitted to tomographic and bronchographic checkups.

1. Etiological Data.—Of these patients 46 were men and 5 were women. Their ages appear in Table I.

TABLE I. AGES OF PATIENTS

AGE	20 or less	20 to 30	30 to 40	40 to 50	50 to 60	60 or over
No. of cases	2	1	8	20	14	4

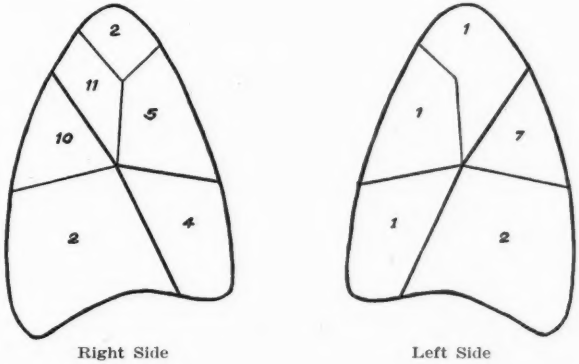
The age was not noted in two cases. A definite etiology was apparent in seven of the fifty-one cases only. In one patient the abscess was subsequent to a sea bath with submersion; in six patients it followed a surgical intervention (two gastrectomies, one prostatectomy, one cholecystectomy, one drained appendicular abscess, and one colon resection for a carcinoma). We have never come across an abscess subsequent to dental avulsion or tonsillectomy.

2. Topographic Data.—Table II shows the location of the suppurating foci we have treated.

TABLE II. LOCATION OF TREATED FOCI

RIGHT SIDE (40)			LEFT SIDE (15)		
Upper lobe 21	Apical s.	2	Upper lobe 4	Apicodorsal	1
	Anterior s.	5		Anterior	1
	Posterior s.	11			
Middle lobe		4	Lingula		1
Lower lobe 12	Apical s.	10	Lower lobe 10	Apical s.	7
	Basal s.	2		Basal s.	2

In fifty-one patients we have found fifty-five suppurative foci; a few patients presenting several foci. Moreover, we have placed in the corresponding lobe without accurately diagnosing the diseased segments some cases of extensive lobar suppuration. Under these restrictions, lung abscess appears in the great majority of cases as a segmental affection.



Right Side Left Side

Fig. 6.—Location of abscesses. We give here only the location of strictly segmental foci.

If we examine the disposition of these segmental foci such as they appear in Fig. 6, we find that the affected segments are, in order of frequency: the posterior segment of the right upper lobe, the apical segment of the right lower lobe, the anterior segment of the right upper lobe (usually affected in its axillary branch), and the middle lobe. As Mattei pointed out, the lung abscess is therefore to be most frequently found on each side of the interlobar fissure.

3. Results of the Treatment.—Table III sums up the therapeutic results.

TABLE III. RESULTS OF CATHETER TREATMENT

I. Cured			
A. Clinically and anatomically completely healed	15		
B. Clinically healed (more than 2 years) Obvious anatomic sequelae	9	64%	72.5%
C. Clinically healed (more than 2 years) No bronchographic and tomographic checkup	9		
D. Recent clinical recoveries (more than 3 months, less than 2 years)	4		
II. Failure of medical treatment	14		27.5%

We have listed under *A* patients who have clinically recovered, in whom good tomographies and bronchographies show a complete *restitutio ad integrum*. Case 5 is of this type.

CASE 5.—F. O. R., a 51-year-old municipal clerk, was an alcoholic patient without any bronchial history. He presented a subacute pneumonitis at the beginning of April, 1949. He was hospitalized on June 1, 1941. X-ray films showed a voluminous opaque focus with a small light spot, in the apical segment of the left lower lobe. Four instillations of one million units of penicillin were given. Lipiodol bronchograph revealed a bronchial short circuit caused by the cavity between the apical and a subapical bronchus sprung from the posterobasal bronchus, quite close to its origin (Fig. 7). After four more instillations (Fig. 8) the patient was radiologically and clinically healed. Final bronchography on July 20, 1949, revealed a strictly normal apical segment. The patient was examined again on Jan. 27, 1951. He was in perfect clinical condition with a normal thoracic image.

Group *B* consists of nine patients whose clinical recovery has remained excellent *at least for two years* after the end of the treatment, but in whom tomographies and especially bronchographies reveal anatomic sequelae—bronchiectasis or residual cavities at the site of the suppurative focus. Such sequelae are very frequent if they are looked for under correct technical conditions but they do not necessarily imply an unfavorable prognosis. Thus we have had under observation for several years some patients with residual cavities that have not become reinfected (we know how numerous are the cases of bronchiogenic cysts which are located in a favorable draining position in the upper lobe and so remain silent) and that have not caused any clinical disturbance in the patients. Easily tolerated and susceptible to a slow spontaneous regression (Fig. 4) such residual cavities do not in themselves require resection. In the preceding section dealing with bronchography, we have described two cases of this type (Cases 2 and 3).

We have grouped in a third category, *C*, nine patients who have been well for more than two years but *for whom no tomographic and bronchographic accurate anatomic checkups have been made*. These patients have all been examined again in the course of 1951; they lead normal lives without any respiratory clinical manifestations; their standard radiologic film is normal or nearly so.

The last group *D* includes patients whose clinical recovery has been complete for *at least three months* but whose treatment took place less than two years ago. What lies in store for these patients who have been treated and who have "recovered" for a little time? For some who present important residual cavities the prognosis is guarded, for others who present relatively unimportant anatomic sequelae we may hope that they will stay clinically healed and will not relapse. Case 6 is an example.

CASE 6.—A. U. D., an unskilled laborer, 31 years old, was without previous respiratory difficulty. On Nov. 7, 1950, he experienced hemoptysis followed by purulent expectoration. The patient was hospitalized on Nov. 17, 1950, and presented a large suppurative focus in the posterior segment of the right upper lobe. After a thirty-four-day treatment of one million units of systemic penicillin and sulfathiazole there was an afebrile, radiologic improvement, yet pus was still raised. The patient was examined by us at the beginning of January, 1951. Bronchography on Feb. 5, 1951, revealed two persisting cavities in the

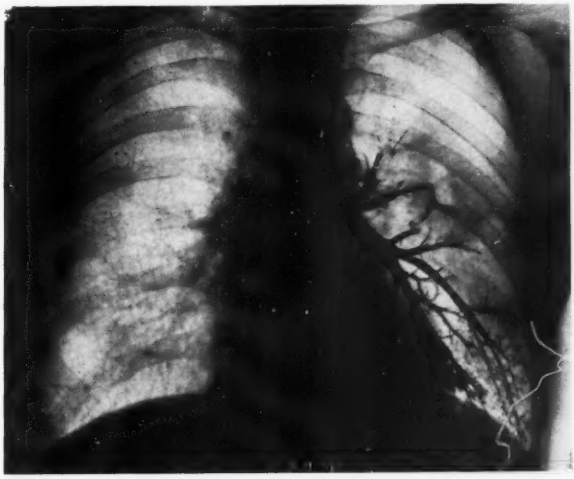


Fig. 7 (Case 5).—Abscess of the apical segment of the left lower lobe. Bronchography of the left lower lobe, bronchial short-circuit between the apical and a subapical bronchus.

Fig. 8 (Case 5).—The Metras's sound in place for instillation.

posterior segment (Fig. 9); icterus was observed. After three months' rest, bronchography on May 7, 1951, indicated spontaneous regression of the residual cavities. The patient was discharged, subject to occasional examinations. Bronchography on Feb. 11, 1953, showed small persisting cavities but a satisfactory clinical condition.

Fig. 9.

Fig. 10.

Fig. 9 (Case 6).—Abscess in the posterior segment of the right upper lobe. Clinical healing. Diodone bronchography at the end of the treatment, two residual cavities.

Fig. 10.—Abscess of the posterior segment of the upper lobe. Initial Diodone bronchography.

Here is a patient who improved under the instillations but who still presented two important residual cavities at the end of the treatment. Should a segmental or a lobal resection be immediately performed? The evolution of these cavities showed that they had a marked tendency toward spontaneous regression. The patient is still submitted to periodical checkups, but we hope that clinical recovery will last without relapse.

In *Group II,* we have listed the fourteen cases in which the medical treatment failed. It forms a rather heterogenous group composed of one patient who is still suffering from a chronic bronchorrhea that can certainly be related to the acute abscess; one patient who died of a brain abscess in spite of the radiologic clearing of the pulmonary lesions; one case in which the lung abscess was just one localization of the multiple suppurative foci of appendicular origin which proved eventually fatal; two cases of chronic suppuration in large residual cavities. The nine remaining patients were submitted to resection, either immediately after a previous cleaning up by means of instillations or following a relapse. Three cases were right pneumonectomies, two right upper lobectomies, two right lower lobectomies, and two left pneumonectomies.

Our statistics therefore show 64 per cent definitely healed, 72.5 per cent if we include those listed under *D* which date back to less than two years. These figures may appear unimpressive, but three facts must be taken into consideration: 1. We have adopted strict criteria of recovery. What would be the results of mere medical treatment or of pneumotomy judged by the same standards? 2. A whole series of our patients came from a thoracic surgeon, on whom a group of doctors call for their worse cases. 3. Last, we have included cases of chronic suppurations (which had gone on for more than three months when the treatment by instillations was begun); in such cases the percentage of medical recoveries hardly reaches 50 per cent, which brings our general statistics down.

If we compare our statistics with the findings published by diverse authors, we find that Mattei obtained 90 per cent recoveries in thirty-three cases of apparently primary acute abscesses. Very encouraging findings were also published by J. Berard,[7] H. Benard,[5] Abel and his associates,[1] Bourgeois,[14] R. Raynaud, R. Castaing,[19] Duroux, P. Oudet,[61] J. Terrasse,[85] M. Fourestier and Le Boucher,[37] R. Petitjean,[65] K. P. Bopp,[12] Mannes,[48] J. A. Pilheu,[68] M. Zaccaria,[91] and D. Vega.[87]

May these findings be considered as an improvement? Undoubtedly so, in our opinion. Indeed there have been lung abscesses *which healed spontaneously.* It is not easy to establish in what percentage in view of the reduced field of many medical statistics and also in view of the fact that such spontaneous "healings" were often published without having gone through the test of time. Archibald wrote that before disappearing one lung abscess was recorded in two or three statistics under the heading "healings." This humorous statement is certainly not entirely devoid of truth. Mattei states in his book that out of 105 patients suffering from lung abscesses who were treated between 1935 and 1945 in his department by means of various methods, only 17, or 15 per cent, really recovered. It does not appear that the percentage of genuine spontaneous healings could be higher than 30 or 40 per cent even in the best series of cases.

The discovery of new antibiotics (Aureomycin, terramycin, bronchocillin) has undoubtedly increased the possibilities of general treatment, but it also has increased the possibilities of local treatment and does not impair the principle of the superiority of local treatment in the case of a suppurative focus.

4. Indications for Surgery.—What we have just said of medical treatment and its failures defines the part we assign to surgery in the treatment of lung abscesses.

When systemic and local antibiotic treatments have proved unable to dry up the suppuration entirely and when the clinical condition is satisfactory but a quite abnormal radiologic picture is still visible (as for instance, when bronchography reveals dislocated bronchi, a considerable or poorly drained residual cavity) the patient should undergo an operation.

At this stage one type of intervention is advisable—resection. All operative drainage of these residual foci would prove illusory; we share J. Mathey's opinion on this point. On the other hand, we do not think that a residual cavity, if it is of small volume and in a good drainage position, constitutes in

itself an indication for resection. We have seen that such cavities were often quite well tolerated and susceptible to spontaneous regression in the course of the years following clinical recovery. If complications set in, such as hemoptysis and especially relapses in situ, we can then always resort to another cleaning up of the suppurative focus and then resection. An imperfect anatomic healing should always be given a chance to undergo the test of time.

REFERENCES

1. Abel, Heuilly, F., Arnould, and Pillot: Résultats statistiques du traitement des suppurations broncho-pulmonaires par sonde de Métras, Sté de Médecine de Nancy, Nov. 22, 1950.
2. Abel, Heuilly, F., Gosserez, Simonin, and Sadoul: Epithélialisation d'une cavité résiduelle d'abcès pulmonaire, Rev. méd. de Nancy **74:** 206, 1949.
3. Allen, C. I., and Blackmann, J. F.: Treatment of Lung Abscess, J. THORACIC SURG. **6:** 146-172, 1936.
4. Aubert, V.: L'exérèse dans le traitement des suppurations parenchymateuses invétérées du poumon, Lyon chir. **41:** 188, 1946.
5. Benard, H., Rambert, P., and Coury, C.: Le traitement des suppurations broncho-pulmonaires par instillations dirigées de pénicilline, Semaine d. hôp. Paris **23:** 2595-2602, 1947.
6. Benda, R.: Le traitement médical des abcés du poumon, Presse méd. **56:** 75, 1948.
7. Berard, J.: Pénicilline ou chirurgie dans le traitement des abcès pulmonaires, Semaine d. hôp. Paris **25:** 510, 1949.
8. Berard, M., Galy, P., and Dumarest, J.: L'épithélialisation secondaire des cavités d'abcès chroniques du poumon, Presse méd. **54:** 234, 1946.
9. Betts, R. H.: Principles in the Management of a Pulmonary Abscess, Am. J. Surg. **54:** 82-98, 1941.
10. Böger, A., and Bopp, K. P.: Die gezielte selektive Bronchographie mit Hilfe der Métrasschen Sonden und deren Verwendung zur endobronchialen Behandlung einiger Bronchial- und Lungenerkrankungen, Ztschr. f. klin. Med. **146:** 203, 1950.
11. Bonarrico, N.: Collasoterapia secundo Forlanini associata alla térapia endobronchiale, bronchoscopotérapia e broncomédicature nell' ascesso polmonare, Riv. Sicil. d. Tub. **5:** 30, 1951. (Abstracted in Poumon **7:** 383, 1951.)
12. Bopp, K. P.: Zur konservativen Behandlung des Lungenabszesses und der Lungengangrän, Neue med. Welt. **1:** 1623, 1950.
13. Bourgeois, P.: Techniques et thérapeutiques en pneumologie, Paris, 1950, L'Expansion scientifique, française, pp. 269-278.
14. Bourgeois, P., Genevrier, R., and Garaix, J. P.: A propos du traitement des abcès du poumon par pénicilline dirigée par voie bronchique, Bull. et mém. Soc. méd. d. hôp. de Paris **64:** 330-331, 1948.
15. Brocard, H., Hanaut, C., Laplanche, C.: Le traitement antibiotique général des abcès du poumon, J. franç. méd. et chir. thorac. **5:** 494-503, 1951.
16. Brock, R. C.: Lung Abscess, Oxford, 1952, Basil Blackwell & Mott, Ltd.
17. Brown and Lincoln: The Fate of Iodised Oil (Lipiodol) in the Lungs, Surg., Gynec. & Obst. **2:** 194, 1928.
18. Brunner, A.: Die Behandlung Lungenabszessresthölen mit Hilfe der Lobektomie, Schweiz. med. Wchnschr. **77:** 630, 1947.
19. Castaing, R.: Le traitement des suppurations pulmonaires par la pénicilline endobronchique, J. méd. Bordeaux **126:** 257-265, 1949.
20. Castex, M. R., and Capedehourat, E. L.: Technique de l'injection intrapulmonaire transthoracique en thérapeutique, J. franç. méd. et chir. thorac. **2:** 201, 1948.
21. Chalnot, M.: Etat actuel du traitement chirurgical des abcès pulmonaires, Semaine hôp. Paris **26:** 182, 1950.
22. Charpin, J., Metras, H., and Gaillard, C.: Bronchographie dirigée à la Diodone, Presse méd. **59:** 1391, 1951.
23. Jackson, Chevalier L., and Judd, A. R.: The Role of Bronchoscopy in the Treatment of Pulmonary Abscess, J. THORACIC SURG. **10:** 179-200, 1940.
24. Clerf, L. H.: The Bronchoscopic Treatment of Bronchiectasis and Lung Abscess, Am. Rev. Tuberc. **24:** 605-611, 1931.
25. Cottenot, P.: Technique et indications des injections de Lipiodol pour le radiodiagnostic des affections des voies respiratoires, Arch. méd. chir. de l'App. Resp. **6:** 97, 1931.
26. Curtillet, E., Huguenin, A., and Morere, P.: Contribution au traitement des abcés du poumon par injections transpariètales intra-cavitaires de pénicilline, Algérie méd. **53:** 25-34, 1949.

27. Cutler, E. C., and Gross, R. E.: Nontuberculous Abscess of the Lung, J. THORACIC SURG. **6:** 125-155, 1936.
28. Delie, P., and Sourdat, P.: Utilisation des produits iodés hydro-solubles pour la bronchographie, Presse méd. **58:** 1156, 1950.
29. DiGuglielmo, L., and Gaudieri, M.: L'ascesso pulmonare quale malattia ad estenzione zonale, Progr. med., Napoli, April 30, 1948, p. 229.
30. DiRienzo, S.: Exploracion radiologica del bronquio, ed. "El Aténéo" Buenos Aires, 1943.
31. Drouet, P. L., Faivre, G., deRen, G., and Sadoul, P.: Les cavités sèquelles des abcès du poumon, Semaine hôp. Paris **26:** 374-379, 1950.
32. Drouet, P. L., Faivre, G., deRen, G., and Sadoul, P.: Le traitement des abcès du poumon par les injections transpáriètales de pénicilline, Semaine hôp. Paris **26:** 359-368, 1950.
33. Fisher, F. K.: La bronchographie à l'huile iodée: un procèdé de diagnostic nocif, J. Suisse de méd. **80:** 73, 1950.
34. Fisher, F. K.: La reproduction radiologique de l'arbre bronchique au moyen d'un produit de contraste hydrosoluble, J. Suisse méd. **80:** 1035, 1948.
35. Fisher, F. K., and Mully: Contribution à la technique de la bronchographie à L'ioduron B, produit de contraste soluble dans l'eau, J. Suisse méd. **80:** 1033, 1948.
36. Fisher, F. K.: Technique, indication et résultats de la bronchographie à l'Ioduron B, substance de contraste hydrosoluble visqueuse, J. Suisse méd. No. 80, 1950.
37. Fourestier, M., and Le Boucher, C.: Bases rationnelles du traitement médical endobronchique des abcès du poumon, Presse méd. **55:** 822, 1947.
38. Gaensler, E. A., Beakey, J. F., and Segal, M. S.: Relative Effectiveness of Parenteral, Intratracheal, and Aerosol Penicillin in Chronic Snppurative Disease of the Lung, J. THORACIC SURG. **18:** 546, 1949.
39. Galy, P., and Delarue, J.: Etude anatomique, classification, origine et histogènèse des "kystes aériens" du poumon, J. franç. méd. et chir. thorac. **5:** 519, 1951.
40. Garcin, C.: Suites cliniques et radiologiques éloignées des abcès du poumon traités par pénicilline endobronchique, Thèse Marseille, 1952.
41. Guerrichio: Trattamiento delle suppurazione polmonare con iniegoni transtoraciche di penicilline, Riforma Medica **8:** 89-93, 1947.
42. Hellstrom, B., and Holmgren, H. J.: Réactions du poumon à la bronchographie aux substances hydrosolubles (Umbradyl). Etude expérimentale sur l'animal, Acta Radiol. **32:** 5, 1949.
43. Huizinga, Elleo, and Smelt, G. J.: Bronchography, Assen, Netherlands, 1949, Van Gorgum.
44. Ioannou, J.: Aspect bronchographique de quelques territoires pulmonaires, Presse Méd. **57:** 1208, 1949.
45. Iselin, M., Sureau, B., and Desmot, R.: Pénicilline et traitement chirurgical des abcès pulmonaire, Presse méd. **55:** 478, 1947.
46. Kourilsky, R.: L'injection de Lipiodol dans les abcès du poumon, Arch. méd. chir. de l'Appl. Resp. **6:** 127, 1931.
47. Lemoine, J. M., and Rose: 90 abcès du poumon traités par broncho-aspiration, J. franç. méd. et chir. thorac. **2:** 432, 1948.
48. Mannes, Priest, and Tixhon: Etude des sèquelles radiologiques des abcès soignés par pénicillothérapie endobronchique, J. Belge de Radiologie **33:** 44-73, 1950.
49. Martin, P. L.: Conception actuelle de la bronchographie, Thèse Bordeaux, 1949.
50. Mannes and Tixhon: Bulle aèrienne sèquellaire d'abcès, J. franç. méd. chir. thorac. **2:** 579-582, 1948.
51. Marini: La penicillina nella dura degli ascessi polmonari, Lotta contra la Tub. **5:** 383, 1946.
52. Mattei, C., Recordier, M., Metras, H., and Barbe, A.: Essai de traitement des suppurations pulmonaires par les instillations endobronchiques de pénicilline, Presse méd. **54:** 185-186, 1946.
53. Mattei, C., Tristani, M., and Barbe, A.: Pénicillothérapie endobronchique précise des abcès du poumon et les lésions pulmonaires en foyer, Paris, 1948, Masson et Cie, Vol. I.
54. Metras, H., Gregoire, M., Lieutier, J., and Gaillard, C.: Quelques problèmes posés par la pénicilline endobronchique dans le traitement des abcès du poumon, Presse méd. **58:** 471, 1948.
55. Metras, H.: Etude bronchographique des résultats de la pénicilline endobronchique dans les abcès pulmonaires, Soc. France de Path. Resp., Feb. 13, 1949.
56. Metras, H.: Le cathétérisme des bronches lobaires avec une sonde en caoutchouc. Son intérêt pour l'injection de Lipiodol et l'aspiration bronchique, Presse méd. **52:** 181, 1944.
57. Meyer, Coudaud, and Ribierre: Formations kystiques aériennes apparues après un abcès du poumon, Bull. et mem. Soc. méd. hôp. Paris **23:** 751, 1947.

58. Morales, O., and Heiwinkel, H.: Une substance de contraste visqueuse soluble dans l'eau. Rapport préliminaire, Acta Radiologica. Schwd. **30:** 317, 1948.
59. Morales, O.: Nouvelles études avec l'Umbradyl viskos, Acta Radiologica. Schwd. **32:** 317, 1949.
60. Neuhof, H., and Tourof, A. S. W.: Acute Putrid Abscess of the Lung, Surg., Gynec. & Obst. **63:** 353-368, 1936; **66:** 836-857, 1938.
61. Oudet, P.: A propos du traitement des abcès du poumon, Soc. méd. de Strasbourg, Jan. 29, 1949 and Feb. 26, 1949.
62. Oudet, P.: Exploration des cavités pulmonaires par les produits de contraste hydrosolubles pour bronchographies, J. franç. méd. et chir. thorac. **5:** 309, 1951.
63. Overholt, R. H., and Rumel, W. R.: Factors in the Reductions of Mortality From Pulmonary Abscess, New England J. Med. **224:** 441-454, 1941.
64. Perron, M.: Quelques observations d'abcès du poumon traités par la Pénicilline endobronchique, Lyon méd. **51:** 852-856, 1947.
65. Petitjean, R.: Traitement médical et chirurgical actuel des abcès aigus du poumon, Thèse Strasbourg, 1950.
66. Pickering, D., Grenville, R., and Mathers: Local Instillation of Penicillin in Lung Abscess, Lancet **17:** 530, 1945.
67. Pieri, J., and Casalonga, J.: Sonde à extrèmité terminale orientable pour cathétérisme des bronches, Presse méd. **58:** 1010, 1949.
68. Pilheu, J. A., and Leston, J. M.: Supuraciones pulmonares tratadas con la Sonda de Metras, El Dia Medico, Buenos Aires **72:** 2868, 1949.
69. Pruvost, P.: Abcès du poumon, ed. 2, Paris, 1945, Encylopédie Méd. Chir.
70. Reboul, J., Martin, P. L., and Castaing, R.: Importance de la bronchographie dans l'étude des abcès du poumon, Jour. de radiologie et d'Electrologie et Arch. d'électricité Méd. Paris **31:** 3, 1950.
71. Ritvo, M.: Chest X-Ray Diagnosis, Philadelphia, 1951, Lea & Febiger.
72. Robertson, P. W., and Forgan Morle, K. D.: Delayed Pulmonary Complications of Bronchography, Lancet **1:** 387, 1951.
73. Rosenthal, F.: Intrabronchial Penicillin for Lung Abscess, Lancet 829-830, 1947.
74. Santy, P., Berard, M., Galy, P., and Dumarest, J.: A propos de l'origine acquise de certains kystes pulmonaires bronchiques de l'adulte, Revue "Le Poumon" **1:** 7, 1946.
75. Santy, P., and Berard, M.: Les résultats du traitement chirurgical dans l'abcès du poumon, Presse méd. **54:** 15, 225, 1944.
76. Scarinci, C., and Ciamillo, E.: Diagnostica broncografica e terapia endobronchiale guidata negli ascessi polmonari acuti del lobo superiore destro, Riforma Medica, 1951.
77. Scarinci, C.: Broncografica e zone del polmone, Archivi di Tisiol. **5:** 943, 1950.
78. Scarinci, C.: I recenti progressi della broncografiaesequita con Ioduron B prodotto di contrasto idrosolubile, Radiologica Medica **36:** 12, 1950.
79. Sergent, E.: Suppurations bronchiques, pulmonaires, pleurales et médiastinales, Paris, 1940, Masson et Cie.
80. Shaw, Robert R., and Paulson, Donald L.: Pulmonary Resection for Chronic Abscess of the Lung, J. THORACIC SURG. **17:** 514, 1948.
81. Sicard and Forestier: Diagnostic et thérapeutique par le Lipiodol, Paris, 1928, Masson et Cie.
82. Sloan, J. Q., Bain, J. P., and Brucer, M. M.: Depth of Penetration of Nebulized Substances in the Respiratory Tree, Proc. Soc. Exper. Biol. & Med. **66:** 264, 1947.
83. Stivelman, B. P., and Kavee, J.: Treatment of Acute Putrid Lung Abscess With Penicillin and Sulfadiazine, Ann. Int. Med. **30:** 343-363, 1949.
84. Theilaud, G.: Contribution à l'étude des sèquelles des suppurations pulmonaires aiguës primitives, Thèse Bordeaux, 1949.
85. Terrasse, J., Mory, F., Lere, S., and Thevenet, L.: Sur 57 cas de suppurations pleurales et broncho-pulmonaires traitées par la Pénicilline, Bull. et mém. Soc. méd. hôp. Paris **64:** 326, 1948.
86. Trocme, C., Brien, and Becoin: Sèquelles tardives des abcès pulmonaires. Exploration lipiodolée, Soc. Franç. Path. Resp. Feb. 12, 1950 (In J. Franç. méd. et chir. thorac. **4:** 459-463, 1950).
87. Vega, D. D., and Aseda, G. M.: Applicaciones de la Sonda de Metras, Rev. Tuberc. Uruguay **17:** 197-205, 1949.
88. Weisel, W., Raine, F., and Owen, J. C.: Acute Lung Abscess, Am. Rev. Tuberc. **61:** 474-481, 1950.
89. Wood, and Grath: Management of Pulmonary Abscess, Surgery **20:** 187, 1946.
90. Young, E. H., and Gantz, E.: The Persistence of Lipiodol in the Lungs Following Bronchography, Brompton Hospital Reports **10:** 107, 1941.
91. Zaccaria, M., and Marcassa, S.: Traitement des abcès, Minerva méd. **38:** 454-455, 1947.

BRONCHOSPIROMETRY WITH THE CARLENS DOUBLE LUMEN CATHETER

Evaluation and Exercise Test

K. Inada, M.D.,* S. Kishimoto, M.D.,** A. Satô, M.D.,** and T. Watanabe, M.D.***
Okayama, Japan

INTRODUCTION

ALTHOUGH it is well recognized that bronchospirometry is a necessary and valuable method for the study of respiratory physiology and for the determination of operative indication and the extent of operative involvement in ordinary chest surgery, it is surprising that it is not popular. This is probably due to its technical difficulties and also the general fear that it may cause not a little suffering to the patient. Since Jacobaeus a number of reports on the bronchospirometric method have been published. Many workers have tried to modify the technique in order to make the procedure less trying for the patient.

We have performed bronchospirometry on about 200 tuberculous patients as a routine examination before thoracic operations during the two years from April, 1950, to April, 1952, at National Okayama Sanatorium. The bronchoscope, which we used in this series, was a flexible metal single lumen bronchoscope modified by Urabe and his associates.[1] It was a Norris-type catheter, equipped with mouthpiece and nose clip instead of the face mask.

As we gained added experience the following disadvantages of this catheter were noticed: 1. There were often difficulties in introducing the catheter into the left main bronchus because of the friction caused by the external rubber coat; this was especially true among women patients. 2. A tear often occurred in this rubber coat during introduction. 3. Aspiration of secretions was difficult. 4. In one patient, whose right lung function was moderately impaired, the value recorded as much less as was actually estimated. 5. The use of a mouthpiece and nose clip makes the patients feel suffocated and causes labored breathing.

As a result, successful recordings were obtained only in about one quarter of the total number of persons examined.

In 1949 Carlens[2] described a new type of a double lumen catheter and reported good results. In June, 1952, we received this catheter; at the present

From the Department of Surgery, the Okayama University School of Medicine and the Surgical Service of the National Okayama Sanatorium, Okayama, Japan.
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*Lecturer in Surgical Clinic at Okayama University School of Medicine.
**Thoracic surgeon at National Okayama Sanatorium.
***Assistant surgeon at Okayama University School of Medicine.

time it has been used in almost 100 cases and has been found to fulfill all the requirements of such an instrument. Because we have actually recognized its superiority to ordinary old bronchospirometry and since very little literature has been available since Carlens' original article, I think it is important to report our experiences with this catheter.

TECHNICAL PROCEDURE

The premedication and topical anesthesia are approximately the same as those employed for bronchoscopy. Half an hour before the procedure, a hypodermic injection of 60 to 100 mg. of morphine hydrochloride and 0.5 mg. of atropine sulfate is administered. Anesthesia of the throat and larynx is secured by the spraying of 5 per cent cocaine hydrochloride; the trachea and main bronchus are then anesthetized by fractional instillation of about 2 ml. of the same solution. We use a Gebauer-type twinspirometer with the tanks containing pure oxygen. In most cases the catheter is very easily introduced under mirror guidance, even without the use of stylet and without tying the rubber hook as Carlens describes. We have not as yet encountered an instance in which introduction of the catheter was impossible. In this regard the catheter is much superior to an ordinary old bronchoscope. It is very easy to feel when the catheter is introduced into the correct position. Carlens states, "It is provided with a little hook that is *automatically* engaged by the carina." It is really like that. When it is once introduced into the correct position and fixed by the inflation of two cuffs, it scarcely slips by a moderate movement of the body or changing of its position. Therefore examinations such as exercise test and those made in different positions are easily carried out.

In most cases the test was smoothly performed for forty minutes without any discomfort to the patient. After examination the patient always stated that he had experienced less pain than by bronchoscopy; also the medical students, on whom the tests to be described were made, said they were able to breathe more easily than in ordinary external spirometry.

There is no doubt that the catheter has the following features of superiority: 1. It requires no skilled technique and no complex equipment. 2. Introducing the catheter is easy, and it can be easily placed in the correct position. 3. The tests made during exercise and in different positions can easily be carried out. 4. It is much less trying for patients than the ordinary old bronchoscope. 5. Sputum can be easily aspirated.

COMPARISON OF CARLENS TUBE AND FLEXIBLE SINGLE LUMEN METAL BRONCHOSCOPE

It is well known that in bronchospirometry there is an increase in respiratory frequency and minute ventilatory volume, and a consequent increase in oxygen consumption, with the corresponding decrease in vital capacity and maximum breathing capacity. This is brought about by the strain and apprehension of the patient at the examination, the effects of the local anesthetic, and the modification of the breathing mechanism which causes the patient to breathe through a stenosis. Urabe[1] reported an average increase of 35.6 per cent in minute volume and 29.7 per cent increase in

TABLE I. COMPARISON OF SUMMATION OF OXYGEN CONSUMPTION AND VITAL CAPACITY FROM BOTH LUNGS BY FLEXIBLE SINGLE LUMEN METAL BRONCHOSCOPE AND BY ORDINARY SPIROMETRY IN 10 TUBERCULOUS PATIENTS

	OXYGEN CONSUMPTION (C.C.)				VITAL CAPACITY (C.C.)			
CASE	RIGHT	LEFT	SUM.	$\frac{R+L}{S}$ (%)	RIGHT	LEFT	SUM.	$\frac{R+L}{S}$ (%)
1	160	200	308	116.8	1040	1830	3030	94.7
2	230	100	220	150.0	1350	870	2300	96.5
3	80	230	250	124.0	460	1120	1770	89.2
4	330	40	300	123.3	2000	230	2250	99.1
5	300	150	276	163.0	900	580	1550	94.8
6	360	130	350	140.0	2250	1320	3620	98.5
7	300	77.3	225	165.8	880	300	1390	84.8
8	45	220	157	162.4	1440	120	1420	107.6
9	255	180	350	124.2	1640	1070	2900	93.4
10	220	100	330	96.9	660	1420	2000	104.0
Average				133.6				92.1

S—Vital capacity obtained by ordinary spirometry.

oxygen consumption and an average decrease of 9.2 per cent in vital capacity and of 21.9 per cent in maximum breathing capacity in ten healthy male adults when a flexible single lumen metal bronchoscope was used. Suzuki[3] found an average increase of 32 per cent in oxygen consumption and an average decrease of 20.0 per cent in vital capacity in sixteen healthy adults using the double lumen metal brochoscope which he designed. In our series, increase in oxygen consumption and decrease in vital capacity were 33.6 per cent and 7.9 per cent on the average, respectively, in the ten tuberculous patients examined with the flexible single lumen metal bronchoscope (Table I). If we take the increase in oxygen consumption as an index of strain on the patient and the decrease in vital capacity as an index of stenosis breathing and compare the Carlens tube with the old bronchoscope in regard to these two points, it is self-evident that the former is more physiologic than the latter.

As Table II shows, in the test on the healthy medical students the increase in oxygen consumption and the decrease in vital capacity average 9.3 per cent and 0.7 per cent, respectively. As Table III shows, in the test made on

TABLE II. COMPARISON OF SUMMATION OF OXYGEN CONSUMPTION AND VITAL CAPACITY FROM BOTH LUNGS BY CARLENS TUBE AND BY ORDINARY SPIROMETRY IN 10 STUDENTS

	OXYGEN CONSUMPTION (C.C.)				VITAL CAPACITY (C.C.)			
CASE	RIGHT	LEFT	SUM.	$\frac{R+L}{S}$ (%)	RIGHT	LEFT	SUM.	$\frac{R+L}{S}$ (%)
1	165	150	320	98	1910	1710	3650	99.1
2	180	180	310	116	2010	1740	3800	98.7
3	155	155	285	109	2170	2100	4250	100.4
4	187	95	255	110	1610	1490	2940	105.4
5	113	156	260	103	2040	1760	3480	109.0
6	165	205	330	112	1590	1540	3780	82.8
7	190	185	345	108	1530	1490	3050	99.0
8	205	185	315	123	1950	1840	3960	96.0
9	115	105	262	84	1830	2010	3760	102.1
10	240	167	438	108	2160	2030	4080	102.7
Average	174	167	312	109.3	1880	1779	3675	99.3
	R:L 51.1:48.9				R:L 51.4:48.6			

S—Vital capacity obtained by ordinary spirometry.

TABLE III. COMPARISON OF SUMMATION OF OXYGEN CONSUMPTION AND VITAL CAPACITY FROM BOTH LUNGS BY CARLENS TUBE AND BY ORDINARY SPIROMETRY IN 10 TUBERCULOUS PATIENTS

	OXYGEN CONSUMPTION (C.C.)				VITAL CAPACITY (C.C.)			
CASE	RIGHT	LEFT	SUM.	$\frac{R+L}{S}$ (%)	RIGHT	LEFT	SUM.	$\frac{R+L}{S}$ (%)
1	110	220	265	124.5	570	820	1420	97.8
2	200	103	205	147.8	820	530	1440	90.0
3	210	90	280	103.5	1070	460	2080	73.5
4	35	160	140	139.2	140	1160	1370	94.8
5	105	155	205	126.8	510	690	1290	93.0
6	100	200	308	97.4	920	930	1920	96.3
7	230	55	245	116.3	280	1540	2050	88.7
8	185	90	265	103.7	1660	650	2190	105.4
9	120	215	318	105.3	680	960	1430	114.6
10	60	180	255	94.1	310	1030	1350	99.2
Average				113.7				94.8

S—Vital capacity obtained by ordinary spirometry.

tuberculous patients these figures are 13.7 per cent and 5.2 per cent, respectively. This demonstrates that the test with Carlens tube can be carried out on the diseased as well as on the healthy person. From this fact the superiority of the Carlens tube is well understood. The difference in the increase of oxygen consumption during bronchospirometry is marked, revealing that this procedure is much less trying for the patient than any older method.

BRONCHOSPIROMETRY DURING EXERCISE

Although bronchospirometry is a more advanced method than the usual spirometry as a test of lung function, it shows only a static value as in the case of the latter. It is not only useful for the study of important physiologic questions, but it is also necessary in practical medicine to be informed of the dynamic function of each lung and the adaptability of each lung to physical strain.

Following Carlens' example[4] we have constructed a cyclergometer, on which the subject is able to pedal in the supine position. The test is made in the following manner: During the first five minutes an ordinary bronchospirometry is carried out in the supine position. Then the patient continuously pedals the ergometer at the speed of sixty rotations a minute for three minutes in connection with the spirometer; at the same time a tracing is recorded. Exercise tests have been made in this manner on fifteen tuberculous patients. Principally, the oxygen intake was calculated from the tracing at rest and during exercise and the figures for the oxygen intake for each lung during exercise were presented in percentage of the corresponding figures at rest.

As Table IV shows, the increase of oxygen intake during exercise test in the worse lung varies between 240 per cent and 493 per cent, averaging 350.9 per cent; in the better lung the result was 197 per cent, 455 per cent and 351.6 per cent, respectively, in comparison with the basal values. This means that the average increase rate of oxygen consumption during exercise is equal in both the worse and the better lung. It was expected that the lung, which was inferior to the other because of tuberculous involvement and because of its affected function as demonstrated by bronchospirometry at rest, would show

TABLE IV. COMPARISON OF DIFFERENTIAL OXYGEN CONSUMPTION FOR EACH LUNG AT REST AND DURING EXERCISE (C.C.)

CASE	AT REST			DURING EXERCISE		
	RIGHT	LEFT	R : L	RIGHT (%)	LEFT (%)	R : L
1	107	210	34 : 66	300 (280)	613 (292)	33 : 67
2	25	257	9 : 91	60 (240)	840 (326)	7 : 93
3	115	345	25 : 75	510 (443)	850 (247)	37 : 63
4	210	82	72 : 28	500 (238)	200 (244)	71 : 29
5	105	173	38 : 62	320 (304)	630 (364)	34 : 66
6	170	190	47 : 53	468 (292)	572 (301)	45 : 55
7	180	210	46 : 54	590 (327)	675 (321)	47 : 53
8	147	167	47 : 53	320 (217)	330 (197)	49 : 51
9	198	110	64 : 36	693 (350)	360 (372)	66 : 34
10	173	130	57 : 43	710 (408)	500 (384)	59 : 41
11	215	98	69 : 31	660 (307)	280 (285)	70 : 30
12	160	180	47 : 53	448 (280)	540 (300)	45 : 55
13	130	180	42 : 58	640 (493)	820 (455)	44 : 56
14	160	100	62 : 38	590 (380)	380 (370)	61 : 39
15	233	105	69 : 31	870 (373)	470 (438)	65 : 35
Average	155	170	47.7 : 52.3	512 (330)	537 (316)	48.8 : 51.2

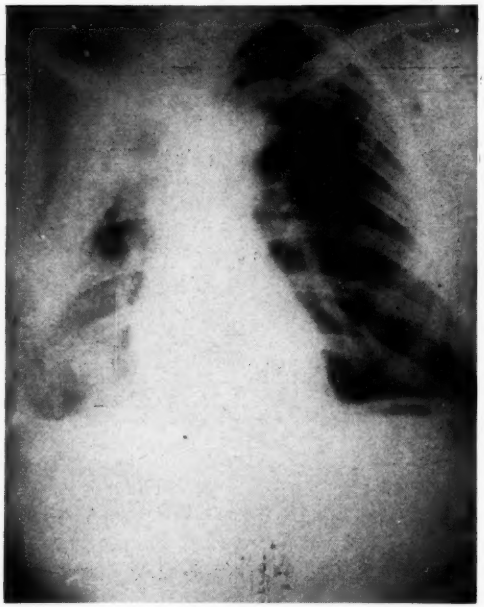
The numbers in parentheses indicate the rate of increase of oxygen consumption in percentage.

relatively smaller increases in the oxygen consumption during exercise than the healthier lung, but that was not the case. We were surprised to find that the part played by each lung did not change beyond the limits of the error of the method during work as compared with the rest values. (Case 2 in Table IV is a good example.)

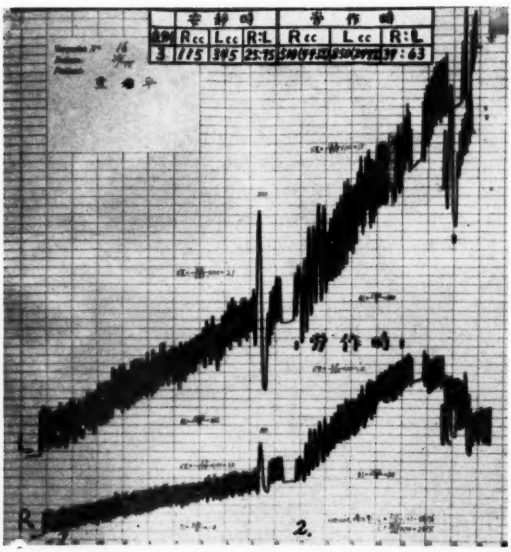
Therefore as Carlens pointed out the theory that the function of the inferior lung approaches its maximum limit of capacity with regard to oxygen intake closer, even during rest, than the healthy lung cannot be correct. But the ventilatory equivalent of the superior lung shows a tendency to slightly more favorable values during exercise as compared with that of the inferior lung (Table V). The inhalation of pure oxygen being, of course, not physiologic, more detailed research is necessary. Two cases are illustrated (Figs. 1 and 2).

TABLE V. COMPARSION OF VENTILATION EQUIVALENT FOR EACH LUNG AT REST AND DURING EXERCISE

CASE	AT REST		DURING EXERCISE	
	RIGHT	LEFT	RIGHT	LEFT
1	3.3	1.9	3.6	1.8
2	3.8	1.7	5.3	0.91
3	3.3	2.1	1.4	1.5
4	2.6	4.6	1.4	2.2
5	3.8	2.6	2.2	1.3
6	4.7	4.2	1.9	1.8
7	2.8	2.0	1.7	1.2
8	2.2	1.8	1.8	1.3
9	3.1	4.5	1.1	1.6
10	2.2	2.3	1.2	1.2
11	2.3	1.9	1.2	1.8
12	2.7	2.2	1.8	1.2
13	3.6	3.3	1.1	0.94
14	2.9	4.4	1.7	2.4
15	2.8	6.0	1.4	1.7

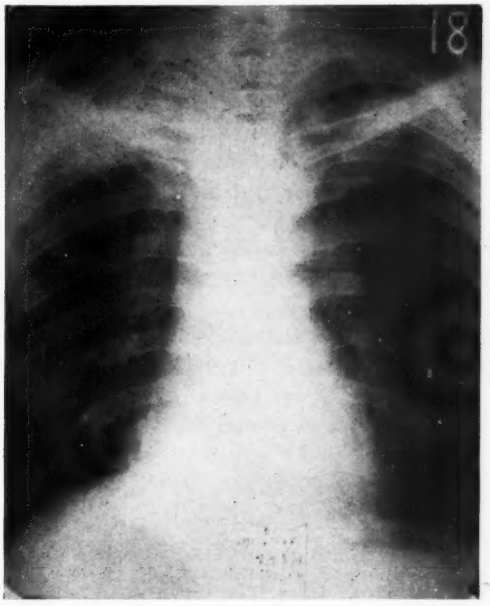


A.

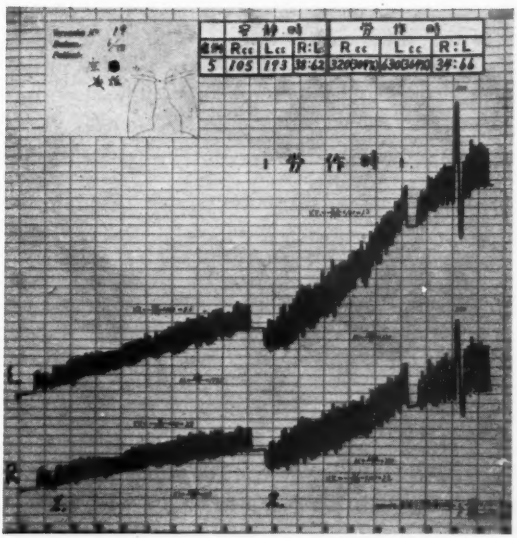


B.

Fig. 1 (Case 3).—*A*, Roentgenogram of the chest. Right-sided postthoracoplasty tuberculosis with moderately thickened pleura. *B*, Bronchospirometric tracing. *R*, right lung; *L*, left lung. *1*, bronchospirometry at rest; *2*, during exercise.



A.



B.

Fig. 2 (Case 5).—*A*, Roentgenogram of the chest. Right-sided apical tuberculosis with cavity and thickened pleura. *B*, Bronchospirometric tracing. *R*, right lung; *L*, left lung. *1*, bronchospirometry at rest; *2*, during exercise.

SUMMARY AND CONCLUSION

1. Experiences with the Carlens double lumen catheter have been described and its superiority emphasized.

2. Using this catheter it was found that the increase in oxygen consumption during bronchospirometry in ten healthy medical students was on the average 9.3 per cent and the decrease in vital capacity was 0.7 per cent.

3. These figures were 11.3 per cent and 5.2 per cent, respectively, in ten patients with pulmonary tuberculosis.

4. It was noted that these same figures were 33.6 per cent and 7.2 per cent, respectively, on bronchospirometry with a modified Norris-type flexible single lumen metal bronchoscope.

5. A new lung function test (bronchospirometry during exercise) and results obtained with fifteen tuberculous patients were described. We agree with Carlens in the following conclusions: (a) The theory that the function of the diseased lung with regard to oxygen intake approaches, even at rest, more closely the maximum limit of its capacity is not correct. (b) It was found that the part played by each lung did not change beyond the limits of the error of the method during work as compared with the rest values.

BRONCHOSPIROMETRY IN DIFFERENT POSITIONS

Studies of the change of lung function in different positions are found in the literature, but they have been made for the most part by a spirometric method. Regarding bronchospirometric study, few articles are available in the literature at present. This is mainly due to technical difficulties. Carlens tube has settled these difficulties.

It is important both for physiologic study and for practical medicine to be informed of the effect of postural changes on the lung function. We have studied this problem on ten healthy medical students by bronchospirometry with the Carlens tube in supine, sitting, and both lateral positions.

TECHNICAL PROCEDURE

Premedication and topical anesthesia was similar to that previously described. The test was done in the following manner: First an ordinary

TABLE VI. DIFFERENTIAL TIDAL AIR (C.C./MIN.) AND PER CENT OF TOTAL

	SUPINE				RIGHT LATERAL				LEFT LATERAL				SITTING			
	VOLUME		%		VOLUME		%		VOLUME		%		VOLUME		%	
CASE	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
1	250	240	51	49	260	210	55	45	200	220	48	52	240	230	51	49
2	380	240	61	39	440	260	63	37	190	360	35	65				
3	360	290	55	45	330	220	60	40	240	260	48	52	400	410	49	51
4	210	200	51	49	200	180	54	46	180	190	48	52	230	220	51	49
5	310	260	54	46	300	280	52	48	280	310	47	53	310	310	50	50
6	540	530	51	49	530	530	50	50	580	570	51	49				
7	300	290	51	49	340	280	55	45	310	320	49	51	410	350	54	46
8	420	410	51	49	400	370	52	48	330	400	45	55	550	510	52	48
9	640	620	51	49	680	630	52	48	680	710	48	52	700	620	53	47
10	580	570	51	49	610	530	54	46	520	570	47	53				
Average	399	365	52	48	409	349	54	46	351	391	47	53	408	380	52	48

bronchospirometry was performed in the supine position for ten minutes, then subsequently in the right lateral and in the left lateral positions for five minutes, respectively, and last the tracing was recorded in the sitting position for ten minutes. The following values can be calculated for each lung from each tracing: tidal air, reserve air, vital capacity, minute ventilation volume, minute oxygen intake, and ventilation equivalent.

RESULTS OBTAINED

1. *Tidal Air (Table VI).*—As Table VI shows, in all but one person the tidal air of the right lung was greater than that of the left lung in the supine position. It was true in the right lateral position in all instances. But in the left lateral position, except in one student, tidal air of the left lung was greater than that of the right lung and it increased in five cases and decreased in five cases, with an average increase of 26 c.c. as compared with that found in the supine position. It is noted, that in the lateral position the tidal air of the under lung was greater than that of the upper lung and showed a slight increase in absolute amount. The tidal air of the right lung increased in five cases and decreased in five cases, with an average increase of 10 c.c. in the right lateral position as compared with the supine. No definite change was found in the sitting position. These differences are not statistically significant ($P > 0.01$).

TABLE VII. DIFFERENTIAL RESERVE AIR (C.C.) AND PER CENT OF TOTAL

	SUPINE				RIGHT LATERAL				LEFT LATERAL				SITTING			
	VOLUME		%		VOLUME		%		VOLUME		%		VOLUME		%	
CASE	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
1	450	410	52	48	330	490	40	60	650	430	60	40	520	480	52	48
2	460	430	52	48	410	470	47	53	920	350	72	28				
3	660	580	53	47	490	720	40	60	870	550	61	39	970	820	54	46
4	440	440	50	50	430	720	37	63	820	390	68	32	700	580	55	45
5	520	480	52	48	500	690	42	58	780	380	67	33	740	720	51	49
6	510	510	50	50	400	340	54	46	500	420	54	46				
7	480	470	51	49	440	570	43	57	670	540	55	45	590	560	51	49
8	510	480	51	49	460	1000	32	68	950	400	70	30	970	950	51	49
9	800	730	52	48	660	810	45	55	910	450	67	33	970	820	54	46
10	790	750	51	49	560	790	42	58	760	460	62	38				
Average	562	528	52	48	468	660	41	59	783	437	64	36	780	704	52	48

2. *Reserve Air (Table VII).*—Reserve air of the right lung was greater than that of the left lung in eight persons while in a supine position. In the right lateral position reserve air of the right lung moderately decreased in all instances, while on the other hand reserve air of the left lung moderately increased. It is reversed in the left lateral position. That is, in the lateral position reserve air of the under lung moderately decreased and that of the upper lung correspondingly increased as compared with the supine. This difference is statistically significant ($P < 0.01$). A moderate increase of reserve air was noted in both lungs when the student was in the sitting position.

TABLE VIII. TOTAL VITAL CAPACITY (C.C.)

CASE	SUPINE	RIGHT LATERAL (%)	LEFT LATERAL (%)	SITTING (%)
1	3080	2220 (72.0)	3000 (97.4)	3120 (100.1)
2	3750	3350 (89.3)	3440 (91.6)	
3	4270	4400 (103.0)	3970 (92.8)	4420 (103.2)
4	3650	3610 (99.0)	3580 (98.1)	3640 (99.5)
5	3800	3790 (99.5)	3780 (99.6)	3930 (103.5)
6	3130	2620 (83.6)	2930 (93.5)	
7	3020	3150 (105.0)	3170 (105.0)	3520 (116.5)
8	3790	3920 (103.3)	3790 (100.0)	4190 (110.0)
9	3740	2930 (78.5)	3590 (96.6)	3740 (100.0)
10	4190	3740 (89.2)	3630 (88.8)	
Average	3642	3370 (93.0)	3488 (96.0)	3794 (105.0)

The numbers in parentheses indicate the percentage as compared with the supine figures.

3. *Vital Capacity (Tables VIII and IX).*—In the right lateral position the total vital capacity of both lungs increased in three cases and decreased in seven cases, with an average decrease of 269 c.c., or 7.4 per cent. In the left lateral position an increase in one instance, a decrease in 8, and no change in another were noted, with an average decrease of 154 c.c., or 4.3 per cent. That is, in the lateral position the total vital capacity slightly decreased as compared with the supine. But this difference was not statistically significant ($P > 0.01$). In the sitting position it increased in five cases and on the average an increase of 173 c.c., or 5 per cent, was observed.

Differential Vital Capacity.—In the supine position, vital capacity of the right lung was greater than in the left lung in all cases and the ratio between both lungs was 52:48. In the right lateral position vital capacity of the right lung was greater than that of the left lung in six cases, but smaller in four cases, and the ratio changed to 51:49. In the left lateral position in eight cases vital capacity of the right lung was greater than that of the left lung and the ratio changed to 51:49. A slight increase was seen in the sitting position. But this difference was not statistically significant ($P > 0.01$). In various positions except the left lateral position vital capacity of the right lung is greater than that of the left lung, but the total vital capacity shows inconstant change.

TABLE IX. DIFFERENTIAL VITAL CAPACITY (C.C.) AND PER CENT OF TOTAL

	SUPINE				RIGHT LATERAL				LEFT LATERAL				SITTING			
	VOLUME		%		VOLUME		%		VOLUME		%		VOLUME		%	
CASE	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
1	1600	1480	52	48	1080	1140	48	52	1580	1420	53	47	1620	1500	52	48
2	2010	1740	54	46	1930	1420	58	42	1900	1540	55	45				
3	2170	2100	51	49	2170	2230	49	51	2050	1920	52	48	2300	2120	52	48
4	1930	1720	53	47	1830	1780	51	49	1880	1700	52	48	1900	1740	52	48
5	2040	1760	54	46	1960	1830	52	48	2100	1680	56	44	1980	1950	50	50
6	1590	1540	51	49	1430	1190	55	45	1500	1430	51	49				
7	1530	1490	51	49	1620	1530	52	48	1480	1690	47	53	1820	1700	52	48
8	1950	1840	52	48	2000	1920	51	49	1950	1840	52	48	2220	1970	52	48
9	1980	1760	53	47	1520	1410	52	48	1850	1740	52	48	2020	1720	54	46
10	2160	2030	52	48	1820	1920	49	51	1560	2070	43	57				
Average	1896	1746	52	48	1736	1637	51	49	1785	1703	51	49	1980	1814	52	48

TABLE X. DIFFERENTIAL MINUTE VOLUME IN LITERS AND PER CENT OF TOTAL

	SUPINE				RIGHT LATERAL				LEFT LATERAL				SITTING			
	VOLUME		%		VOLUME		%		VOLUME		%		VOLUME		%	
CASE	R	L	R	L	R	L	R	L	R	L	R	R	R	L	R	L
1	4.8	4.6	52	48	5.3	4.3	55	45	4.9	4.7	51	49	5.3	5.1	51	49
2	4.6	2.9	61	39	4.4	2.6	63	37	2.7	5.0	35	65				
3	4.7	3.7	56	44	4.6	3.0	61	39	3.1	3.4	48	52	4.8	4.9	49	51
4	4.3	4.1	51	49	4.4	4.0	52	48	3.8	4.0	49	51	4.6	4.4	51	49
5	4.3	3.6	58	42	4.2	3.9	52	48	3.9	4.3	48	52	4.3	4.3	50	50
6	7.5	7.4	50	50	7.4	7.4	50	50	7.5	7.4	50	50				
7	4.2	4.0	51	49	4.9	4.0	55	45	4.7	4.8	49	51	5.8	4.8	55	45
8	6.3	6.1	51	49	6.8	6.3	52	48	5.3	6.4	45	55	8.2	7.6	52	48
9	11.5	11.1	51	49	12.9	12.1	52	48	11.5	12.0	49	51	12.6	11.2	53	47
10	10.4	10.3	50	50	11.9	10.3	54	46	9.6	12.2	46	54				
Average	6.3	5.8	52	48	6.7	5.8	54	46	5.7	6.4	47	53	6.5	6.0	52	48

TABLE XI. TOTAL OXYGEN CONSUMPTION (C.C./MIN.)

CASE	SUPINE	RIGHT LATERAL (%)	LEFT LATERAL (%)	SITTING (%)
1	228	233 (102)	218 (95)	302 (134)
2	360	357 (99)	358 (99)	
3	310	334 (108)	370 (119)	400 (129)
4	340	290 (85)	300 (88)	325 (99)
5	269	275 (104)	268 (100)	288 (107)
6	345	325 (94)	350 (101)	
7	375	382 (102)	325 (87)	382 (102)
8	390	387 (99)	360 (92)	380 (97)
9	440	473 (107)	410 (93)	440 (100)
10	473	459 (95)	430 (91)	
Average	353	352 (100)	338 (96)	360 (107)

The numbers in parentheses indicate the percentage of intake compared with the supine position intake.

TABLE XII. DIFFERENTIAL OXYGEN ABSORPTION IN CUBIC CENTIMETERS AND PER CENT OF TOTAL

	SUPINE				RIGHT LATERAL				LEFT LATERAL				SITTING			
	VOLUME		%		VOLUME		%		VOLUME		%		VOLUME		%	
CASE	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
1	120	108	53	47	133	100	57	43	108	110	50	50	160	142	53	47
2	180	180	50	50	247	110	69	31	165	193	46	54				
3	155	155	50	50	187	147	56	44	170	200	46	54	220	180	55	45
4	170	170	50	50	160	130	55	45	125	175	42	58	180	145	55	45
5	113	156	42	58	150	125	55	45	98	170	37	63	125	163	43	57
6	175	170	51	49	245	80	75	25	160	190	46	54				
7	190	185	51	49	203	180	53	47	155	190	45	55	195	187	51	49
8	205	185	53	47	220	167	57	43	185	175	51	49	205	175	54	46
9	230	210	52	48	313	160	66	34	140	270	34	66	240	200	55	45
10	240	233	51	49	273	186	60	40	150	280	35	65				
Average	178	175	50	50	213	139	61	39	146	195	43	57	189	170	52	48

4. *Minute Ventilation (Table X).*—As Table X shows, a similar tendency is noted as in tidal air.

5. *Minute Oxygen Intake (Tables XI and XII).*—The total oxygen intake of both lungs increased in five students and decreased in five in the right lateral position. But these changes were slight, and there was no change found on the average. In the left lateral position, intake decreased in eight cases and increased in two cases, with an average decrease of 14 c.c., or 4 per

cent. In the sitting position the average increase of 172 c.c., or 5 per cent, was noted.

Differential Oxygen Consumption.—Minute oxygen intake of the right lung was greater than that of the left lung in six students, equal in three, and smaller in one in the supine position. In the right lateral position minute oxygen intake of the right lung (the under lung) was greater than that observed in the supine in all cases but one (Case 4). On the other hand in Case 4, oxygen intake of the left lung showed a further decrease than while in the supine position. It was noted that in the left lateral position oxygen intake of the left lung was greater than the intake of the right lung in all but Case 8. In Case 8 the oxygen intake of the right lung showed a corresponding decrease as compared with the intake in the supine position. It was noted, therefore, that the under lung in the lateral position showed a definite increase in the amount and percentage of the total oxygen consumed as compared with the supine position, and it was reversed in the upper lung. This difference was statistically significant ($P < 0.01$). The right lung in the right lateral position showed a slight but definite increase over the left lateral position.

6. *Ventilation Equivalent (Table XIII).*—The ventilation equivalent of the under lung in the lateral position decreased, and on the other hand it increased in the upper lung over the supine valve. In the right lateral position the right lung showed an average decrease of ventilation equivalent from 3.5 to 3.2; the left lung showed an average increase from 3.2 to 4.7 as compared with the supine position. In the left lateral position the right lung demonstrated a slight increase, and the left lung showed no change. This was due to increased oxygen absorption with relatively less increase in the total air breathed by the under lung. Changes in the ventilation equivalent were considered merely reflections of the change in oxygen absorption and minute ventilation as Rothstein[5] stated.

TABLE XIII. VENTILATION EQUIVALENT

CASE	SUPINE		RIGHT LATERAL		LEFT LATERAL		SITTING	
	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT
1	3.9	4.1	4.0	4.3	4.5	4.3	3.3	3.6
2	2.5	1.6	1.8	2.3	1.6	2.6		
3	3.0	2.3	2.4	2.0	1.8	1.7	2.1	2.7
4	2.7	2.6	2.7	3.1	3.0	2.3	2.5	3.0
5	3.8	2.3	2.8	3.1	4.0	2.5	3.4	2.6
6	4.2	4.3	3.0	9.2	4.7	3.9		
7	2.2	2.1	2.4	2.2	3.0	2.5	2.9	2.6
8	3.0	3.3	3.1	3.7	2.8	3.6	4.0	4.3
9	5.0	5.3	4.1	7.5	8.2	4.4	5.2	5.6
10	4.3	4.4	4.3	5.5	3.4	4.3		
Average	3.5	3.2	3.1	4.3	3.7	3.2	3.3	3.5

DISCUSSION

Among results described two outstanding changes regarding reserve air and minute oxygen intake will be noted. It is well known that the factors that affect the function of lungs are three: conditions of the pleural cavity, the diaphragm, and parenchymal lesions.

Lowys[6] reported that in the lateral position the diaphragm increases its movement, and Fischer[7] stated that in the lateral position the diaphragm of the under side elevates, that of the upper side descends, and the heart and mediastinum shift to the under side.

Akutsu[8] revealed radiologically that in normal persons in the lateral position the diaphragm of the under lung elevates and also increases its movement; the movement of the upper thoracic cage decreases at the same time. He further stated that movement of the diaphragm of the under side is moderately greater than that of the upper side. Rothstein[5] studied bronchospirometry in the recumbent position and found that in the lateral position oxygen intake of the under lung increases; he attributed this increase to an increase of blood flow through the under lung caused by gravitation. In our series the under lung in the lateral position showed a slight increase of tidal air and of minute ventilation, a slight decrease of vital capacity, and a definite decrease of reserve air. This change can probably be attributed to the effect of the movement of the diaphragm. It is apparent that tidal air and minute ventilation increase in the under lung in the lateral position. In addition to this, if the blood flow through the under lung is greater, it is natural that the oxygen intake of the under lung increases. However, it is not known, which is more effective ventilatory or cardiovascular changes; further research is necessary. There is no doubt that the sitting position is less resistant to respiration than other positions.

SUMMARY AND CONCLUSION

1. Bronchospirometry in the supine and both lateral positions has been performed on ten normal persons. Seven of them also had determinations made while sitting.

2. A slight increase in the absolute amount and a definite increase in the percentage of tidal air was noted in the under lung in the lateral position.

3. A definite decrease both in the absolute amount and in the percentage of reserve air was noted in the under lung in the lateral position.

4. Total vital capacity decreased by an average of 7 per cent while lying on the right side and 4 per cent while lying on the left. Differential vital capacity of both under and upper lungs showed a slight decrease as compared with that found while supine.

5. A slight but definite increase both in the absolute amount and percentage of minute ventilation was noted in the under lung in the lateral position.

6. The total oxygen consumption showed no change in the right lateral position and an average decrease of 4 per cent in the left lateral position as compared with while supine. But a definite increase both in the absolute amount and in the percentage of oxygen consumption was noted in the under lung in the lateral position.

7. The ventilation equivalent decreased in the under lung, indicating increased efficiency.

8. A definite increase of oxygen consumption in the under lung in the lateral position is attributable to the increase in minute ventilation and in blood flow through the same lung.

9. In the sitting position tidal air, reserve air, vital capacity, minute ventilation, and total oxygen consumption all increased on the average by 3.1, 36, 5, 3.3 and 7 per cent, respectively. The sitting position was considered less resistant for respiration than other positions.

I wish to express my hearty thanks to Professor S. Tsuda for his guidance.

REFERENCES

1. Urabe, M., Hayashi, S., Kuno, K., and Suga, K.: Study on Bronchospirometry, Jap. J. Thoracic Surg. **3:** 280-286, 1950.
2. Carlens, E.: A New Flexible Double Lumen Catheter for Bronchospirometry, J. THORACIC SURG. **18:** 742-746, 1949.
3. Suzuki, C., Awataguchi, S., Mizuno, S., and Kaishio, K.: Studies on Bronchospirometry, Jap. J. Thoracic Surg. **3:** 217-222, 1950.
4. Björkman, S., and Carlens, E.: The Lung Function During Rest and Exercise in Lung Disease. A Bronchospirometric Study, Acta med. Scandinav. (Supp.) p. 259, 1951.
5. Rothstein, E., Landis, F. B., and Narodick, B. G.: Bronchospirometry in the Lateral Decubitus Position, J. THORACIC SURG. **19:** 821-829, 1950.
6. Lowys, P., Marinet, and Marlin: Étude radiologique de la mécanique pulmonaire dans la position latéro-déclive; son intérêt thérapeutique, Rev. de la tuberc. **2:** 890-909, 1936.
7. Fisher, L.: Effects of Posture on the Diaphragm and Mediastinum, With Special Reference to Phrenicectomy, Am. Rev. Tuberc. **24:** 57-64, 1931.
8. Akutsu, M.: Roentgenkymographic Study on Respiratory Mechanism in Different Position, Kekkaku (Tuberculosis) **18:** 642-670, 1940.

A NEW CATHETER FOR BRONCHOSPIROMETRY WHICH COMBINES THE ADVANTAGES OF THE DOUBLE LUMEN AND SINGLE LUMEN CATHETERS

PIETRO CROCE, M.D.
MILANO, ITALY

ALL the double lumen catheters used for bronchospirometry have the disadvantage of being too obstructive, although this is true in a lesser degree for the Carlens tube.[1] This causes unsatisfactory procedures in many cases. Attempts have been made to substitute single lumen catheters for the double lumen catheters[2, 5] in order that less space may be occupied by the solid structures of the instrument. A strong objection to the single lumen catheter is the variable degree of free space left to the air flow of the right side by the vocal cords; in some instances all passage may be obstructed completely, or almost completely.

I have designed a new catheter for bronchospirometry, which combines the advantages of the double lumen with the single lumen catheter. It consists of a tube 43 cm. in length, destined for the left main bronchus. It bears, at the distal end, an inflatable rubber cuff. Its diameter, in the male type, is 9.2 mm., which gives a sectional area of 67 mm.^{2} The rubber is radiopaque, and only 0.8 mm. thick, which allows a very little loss of space, due to the dead structures of the instrument. At a distance of 12 cm. from the tip, this tube bears a second tube, of a semilunar section, only 6 cm. in length, and of a cross-sectional area of nearly 24 mm.^{2} These measurements have been calculated according to the Fanning equation for the turbulent flows in order to grant the same resistance to the air flow for both lumina:

$$\triangle p = \frac{2 f L V^2}{g D}$$

f = friction coefficient
L = length of the tube
V^2 = square of the velocity of the air flow
g = acceleration of gravity
D = diameter of the tube.

The reduction in diameter of the smaller tube (designed for conducting the air of the right lung) is fully compensated for by the reduction in length. The smaller tube is connected to the larger one, at a point where, when the latter is correctly placed in the left main bronchus, the former is exactly between the vocal cords. Consequently, the small tube, or laryngeal tube, permits an air passage for the right side even in the case of a laryngeal spasm, and it constantly allows the same resistance as the contralateral tube. The small size of the laryngeal tube permits the very large diameter of the longer one.

From Ospedale Sanatoriale "L. Sacco," Vialba, Milano, Italy.
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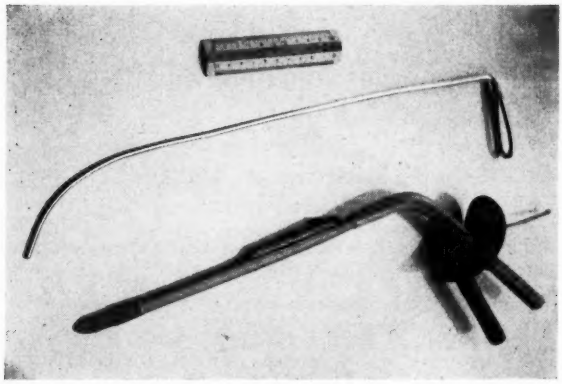

Fig. 1.—The instrument.

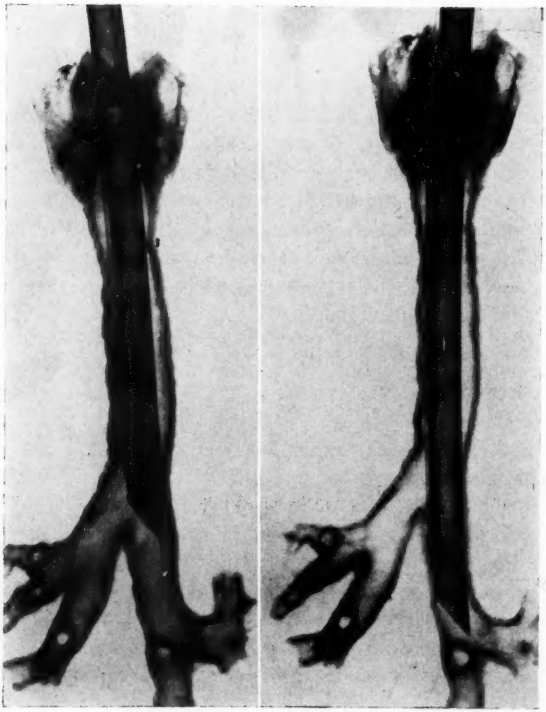

Fig. 2. Fig. 3.

Fig. 2.—The action of the slope in position in the trachea.
Fig. 3.—The catheter in the correct position in the trachea.

Considering the diameter in its functional significance, rather than in its geometric one, it is like having a double lumen catheter made of two equal-sized tubes of 67 mm.^{2} each, that is 134 mm.^{2} totally.

This sectional area is much larger than that of all the other existing tubes for bronchospirometry. In fact, in the Zavod[6] and in the Gebauer[4] tubes, the sum of the sectional areas is about 45 to 50 mm.^{2}; in the Carlens[1] tube, about 80 mm.^{2} in the male size; in the Norris[5] about 45 mm.^{2} for the left, variable for the right; in my^2 single lumen catheter, about 60 mm.^{2} variable for the right.

Moreover, during the intubation with this new catheter, some small spaces remain between the tube and the anatomic formations of the glottis, which provide further space for the air flow of the right side. These spaces are lost with the use of all double lumen catheters. They are all that is conceded to the right side, with the single lumen catheters. In this new catheter, they represent a useful complementary space to be added to the passage already granted by the laryngeal tube.

The air coming from the right lung in the natural way finds, at the level of the larynx, the laryngeal tube, by which it is conducted through the narrow passage of the glottis. At the level of the pharynx, it is again free and, added to the air coming through the small spaces left around the tube, is collected by a properly constructed mouthpiece at the level of the vestibulum oralis.

A further device has been used to direct the catheter almost automatically to the left bronchus. The tip of the catheter is cut at an angle of about 30 degrees. The resulting slope slides on the tracheal carina and directs the tube toward the wanted side.

Though the tube is made of a radiopaque rubber, at the tip of it there is a metal ring which makes the radiopacity perfect and, because of its peculiar form, shows the correct position of the slope (Figs. 2 and 3).

SUMMARY

A new catheter for bronchospirometry is described. The instrument combines the advantages of the double lumen and the single lumen catheters.

REFERENCES

1. Carlens, E.: A New Flexible Double Lumen Catheter for Bronchospirometry, J. THORACIC SURG. **18**: 742, 1949.
2. Croce, P.: Catetere per broncospirometria, Giorn. ital. della Tuberc. **6**: 265, 1952.
3. Gaensler, E. A., Maloney, J. V., Jr., and Björk, V. O.: Bronchospirometry. II. Experimental Observations and Theoretical Considerations of Resistance Breathing, J. Lab. & Clin. Med. **39**: 935, 1952.
4. Gebauer, P. W.: A Catheter for Bronchospirometry, J. THORACIC SURG. **8**: 674, 1939.
5. Norris, C. M., Long, J., and Oppenheimer, M. J.: Bronchospirography: Apparatus and Technique, J. THORACIC SURG. **17**: 357, 1948.
6. Zavod, W. A.: Bronchospirography. I. Description of the Catheter and the Technique of Intubation, J. THORACIC SURG. **10**: 27, 1940-41.

AN UNUSUAL CASE OF BILATERAL DIAPHRAGMATIC HERNIA IN AN INFANT

PAUL F. HAUSMANN, M.D., AND ROBERT K. SALTER, M.D.
MILWAUKEE, WIS.

ALTHOUGH the literature contains numerous case reports dealing with surgical repair of single diaphragmatic defects,[1-5] it was thought worth while to report a case of bilateral diaphragmatic herniae in an infant recently observed at Milwaukee Childrens Hospital, for the following reasons:

1. The child had a short esophagus type of esophageal hiatus hernia, the defect occurring in the right hemidiaphragm.

2. A second defect was accidently produced in the dome of the *left* diaphragm during attempted surgical repair of the hiatus hernia elsewhere. It was this traumatic hernia which was responsible for the major complaints when the infant was seen here.

3. Successful surgical repair of both diaphragmatic defects was eventually accomplished in two stages on a debilitated infant. The first stage was mandatory as a lifesaving procedure.

CASE REPORT

D. K. was first admitted to Milwaukee Childrens Hospital on Dec. 29, 1949, at the age of 4 months. The chief complaint was vomiting of several weeks' duration. The vomiting was associated with abdominal cramps and borborygmi which were relieved by holding the baby in an upright position, and aggravated by horizontal positioning. Belching and flatulence were noted frequently.

The past history revealed that the female infant had been born at another hospital on Aug. 20, 1949. The birth weight was 7 pounds, 12 ounces. Because the child regurgitated feedings frequently, barium studies were done, and a short esophagus type of esophageal hiatus hernia was discovered. An attempt was made at surgical repair of the hernia at the age of 18 days, through a left transthoracic incision. Technical difficulties had made exposure of the esophagus almost impossible, and because of the precarious condition of the infant, the thorax was hastily closed without actual repair of the esophageal hiatus (which of course could not be visualized from the left side), and without lengthening of the esophagus. The baby was discharged from that institution in September, 1949, but returned in November, 1949, because of "cramps," vomiting, and failure to gain weight. There were subsequent bouts of diarrhea. Discharge after the second period of hospitalization was on Dec. 24, 1949, five days prior to admission to Milwaukee Childrens Hospital on Dec. 29, 1949.

Examination on entrance to this hospital revealed a "scrawny," wizened, dehydrated infant weighing 8 pounds, 9 ounces. (This represented a gain of only thirteen ounces in four months.) Skin turgor was poor and the eyes were sunken. There was no respiratory distress or cyanosis. A healed thoracotomy scar was seen on the left side over the course of the fifth rib. Dullness to percussion and diminution of breath sounds were noted over the lower left chest. The heart rate was regular with no audible murmurs.

From the Department of Surgery, Milwaukee Childrens Hospital and Marquette University School of Medicine.
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Initial laboratory data revealed a trace of albumin with occasional clumps of white blood cells in the urine. Leukocytes numbered 12,400 per cubic millimeter, and the hemoglobin value was 12.5 Gm. per 100 c.c. of blood. A chest x-ray film taken on admission (Fig. 1) showed an esophageal hiatus diaphragmatic hernia on the *right* with approximately two-thirds of the stomach above the right diaphragm lying in the cardiophrenic angle. There was bowel in the *left* hemithorax, and pneumonitis in the lung surrounding it. A portion of the left fifth rib had been resected.

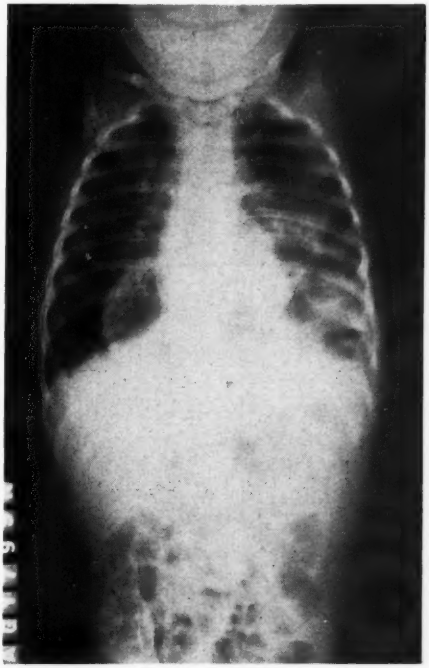


Fig. 1.—Initial chest film showing herniated stomach in the right thorax and colon in the left thorax.

A barium enema substantiated the fact that a portion of the transverse colon lay above the left diaphragm (Fig. 2). Barium studies of the upper gastrointestinal tract revealed that the esophagus was situated in the right hemithorax and that the stomach herniated through an enlarged esophageal hiatus located in the right diaphragm (Fig. 3).

The defects in the two sides of the diaphragm were interpreted as being entirely independent of each other.

Because of the extreme rarity of bilateral diaphragmatic defects in infants, the original films were obtained for comparison. It was seen that at birth the left diaphragm had been intact, but that there was a defect of the right diaphragm which had remained relatively unchanged (Fig. 4).

Surgical exploration on the left was decided upon because it was felt that the cramping, flatulence, diarrhea, and failure to gain weight were referable

to incarceration of the colon, and that correction of this condition should be accomplished first.

First Operation.—Supportive therapy including penicillin for the pneumonitis, and whole blood for the anemia and hypoproteinemia was given to rehabilitate the infant. On Jan. 17, 1950, left thoracotomy was done. A long segment of rib nine was resected subperiosteally for exposure. The roentgen interpretation was corroborated by the discovery that the major portion of the transverse colon had herniated through a small opening in the dome of the left diaphragm. Many adhesions to pleura, lung, and diaphragm were freed

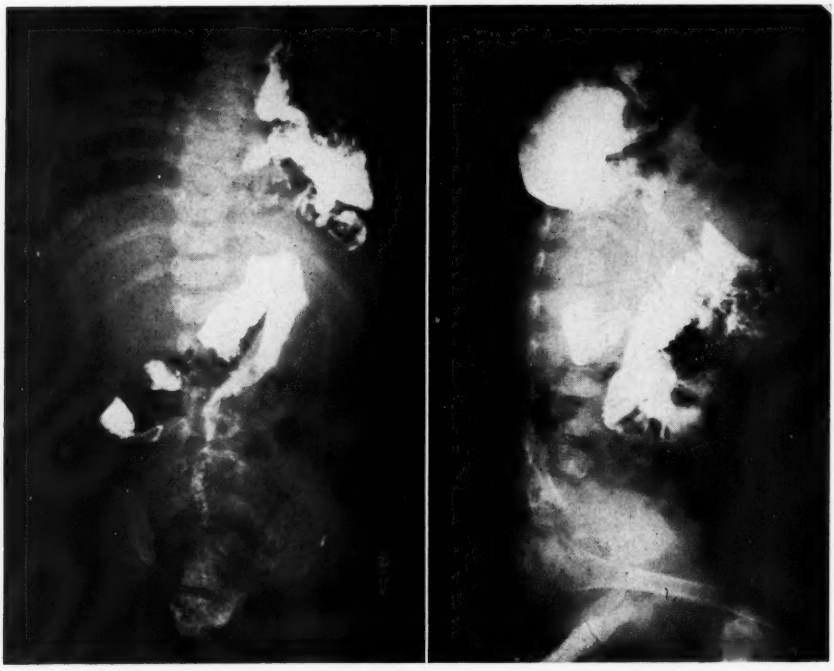
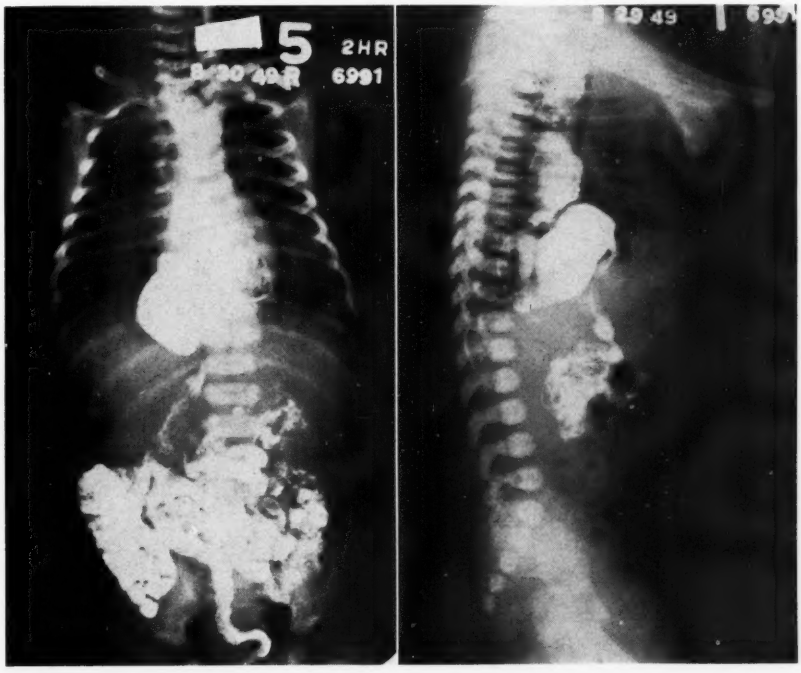


Fig. 2. Fig. 3.

Fig. 2.—Barium enema showing colon in the left thorax.
Fig. 3.—Stomach filled with barium, demonstrating herniation of two-thirds of the organ into the right chest.

before the colon could be returned to the abdomen. The bowel and its mesentery were edematous and purplish in color, but became normally pink upon being freed. There was no hernial sac. The defect, approximately 2 cm. in diameter, was sutured and imbricated with No. A cotton mattress sutures. There was no esophageal hiatus in the left diaphragm, and neither stomach nor esophagus could be visualized from the left approach. The thorax was closed in layers without drainage after re-expansion of the lung. The infant's immediate postoperative condition was only fair with moderate transitory cyanosis for which oxygen therapy was given. The child's temperature rose to 105.2° F. on the evening of the first postoperative day but progress thereafter was satisfactory. Small feedings were given and retained on the second postoperative day.

This hernia was interpreted as traumatic in origin because it was not present on the original films and because of its location in the fibrous dome. This area is not a fusion point, and, as has been pointed out by Harrington,[6] herniation at this point must be regarded as traumatic in origin.



A. *B.*

Fig. 4.—*A*, Original stomach films taken elsewhere showing herniation of the stomach into the right chest but no evidence of herniation of the colon into the left. *B*, Same lateral view.

Fluoroscopy of the stomach and colon performed on Jan. 28, 1950, the tenth postoperative day, showed no change in position of the esophagus and stomach. The colon and small intestine were now seen to be in the abdomen below the diaphragm. The left thorax was clear (Fig. 5). The patient was discharged from the hospital on the fourteenth postoperative day, taking feedings well and gaining weight. There was no respiratory difficulty. Regurgitation of portions of feedings occurred if the child was placed in the prone position immediately after feeding. Bowel function was restored to normal following this stage with disappearance of diarrhea, hyperperistalsis, and anorexia and establishment of one to three normal bowel movements daily. It was deemed advisable to postpone consideration of further surgery until the child had gained weight and strength.

The final admission date was Nov. 15, 1950, when the child was 14 months old. The interval history indicated that the child had done well since the last operation. Multiple small feedings were required to prevent regurgitation. There had been a five-pound weight gain since discharge from previous hos-

pitalization. Physical examination revealed normal findings. Repeat fluoroscopy of the esophagus and stomach showed no change. Because the esophagus was somewhat tortuous, it was felt that it could be sufficiently mobilized to allow placing the stomach entirely under the diaphragm and to allow repair of the diaphragmatic defect.

A. *B.*

Fig. 5.—*A*, Colon x-ray photograph following first operation showing complete reduction of the hernia on the left; colon completely in the abdomen. *B*, Stomach x-ray film following the first operation showing no change in the herniation of the stomach through the right diaphragm.

Preoperative penicillin therapy was given, and on Nov. 21, 1950, under endotracheal gas-ether anesthesia, a right thoracotomy was performed through the bed of the resected ninth rib. There was a free pleural space, and it was seen that at least three-fourths of the stomach, enclosed in a hernial sac, lay above the diaphragm. The sac was incised and the entire circumference of the esophagus freed from the esophagogastric junction to a point 3 cm. proximally. The stomach could then be replaced into the peritoneal cavity. The diaphragmatic defect was closed about the esophagus with interrupted 00 cotton sutures. Mediastinal and diaphragmatic pleurae were utilized to cover raw surfaces and to reinforce the suture line. The lung was re-expanded and the wound closed in layers with interrupted cotton sutures. It was thought that this hernia was definitely congenital in origin.

The postoperative course was uneventful except for vomiting for two days; this was controlled by gastric suction. Penicillin therapy was continued for six days. Feedings of water and formula were begun on the third postoperative

day. Barium swallow on the tenth postoperative day revealed a slightly dilated esophagus with the stomach lying entirely under the diaphragm (Fig. 6). The child was discharged on the seventeenth postoperative day.

Subsequent fluoroscopy in October, 1951, demonstrated that both diaphragmatic defects remain closed. The child has continued to gain weight and is free of symptoms on a normal diet (Fig. 7).

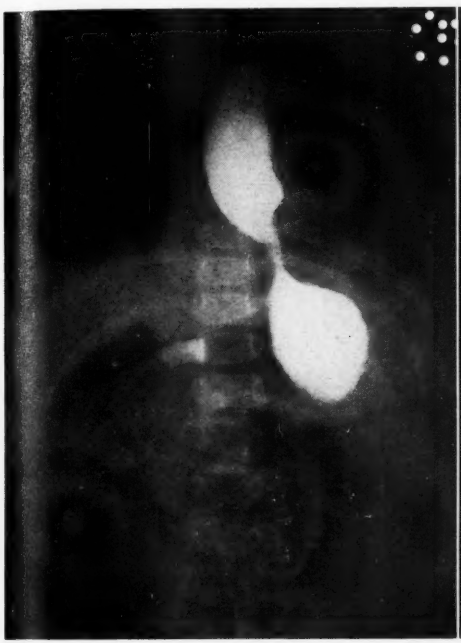


Fig. 6.

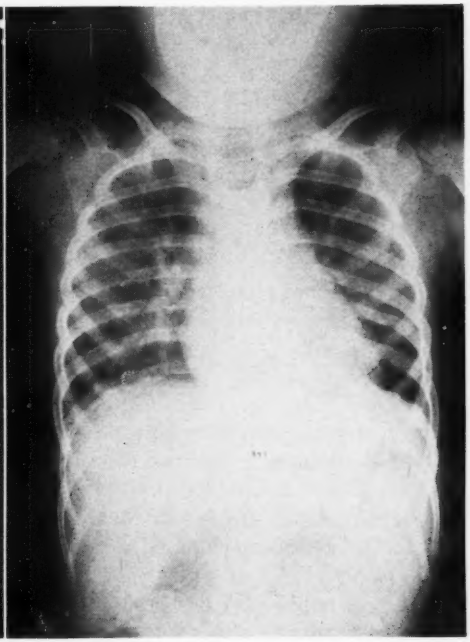


Fig. 7.

Fig. 6.—Stomach x-ray film following second operation showing dilatation of the esophagus but no hiatus hernia.

Fig. 7.—Posteroanterior chest film following second operation showing essentially normal chest.

DISCUSSION

When the infant was first admitted to this hospital the clinical picture was that of partial bowel obstruction, dehydration, and inanition. An initial attempt was made to improve the nutritional status of the patient by careful small oral feedings supplemented by parenteral fluids and whole blood. It became apparent after nineteen days of this type of treatment that definitive surgical therapy was mandatory in spite of the poor condition of the child. Since the symptoms appeared to be primarily referable to incomplete bowel obstruction, and since the radiologist was reasonably certain that there were two diaphragmatic defects, it was felt that correction of the left hemidiaphragmatic defect was most urgent. Following the first stage, there was such a noticeable clinical improvement in the infant, that consideration of surgical repair of the esophageal hiatus was deferred until such time that an appreciable weight gain had taken place, and the child had reached some degree of development commensurate with her age. It was not even established at this time that the esophagus

was of sufficient length to permit placement of the stomach in the abdominal cavity and it was felt that existence of a short esophagus type of esophageal hiatus hernia was not a threat to life, nor near normal development, witness the fact that many adults with this type of deformity lead almost normal lives.

Barium studies were repeated in an attempt to determine whether sufficient length of esophagus could be developed to place the stomach into the abdomen below the diaphragm, and it was finally decided that the tortuosity of the esophagus indicated that such length could be developed. Continued regurgitation of formula, although small in amount, was of sufficient severity in our judgment, to warrant an attempt at repair of the defect.

It has always been our conviction that the transthoracic approach is preferable to the abdominal route in all but foramen of Morgagni types of diaphragmatic herniae. In this particular instance sharp dissection was required bilaterally to free the bowel and stomach from the lung sufficiently to allow replacement of these organs into the abdominal cavity. This was accomplished easily under direct vision, whereas we believe such dissection would have been difficult from an abdominal approach. This was particularly true on the left side since the defect was small and exposure from the abdominal side would have necessitated enlarging the defect considerably. On the right side, mobilization of the esophagus was also easily accomplished under direct vision in contradistinction to the blind traction which would have been necessary from an abdominal approach, particularly since mobilization and retraction of the right lobe of the liver would have been required.

The failure of the child to gain weight demonstrates the necessity for early repair when bowel is contained in the defect. In this case the vascular supply to the colon was impaired as evidenced by thickening and edema of the bowel wall which contributed to the degree of obstruction of bowel lumen.

Careful radiographic studies are essential to determine the type of defect and amenability to surgical repair. In this instance such studies established first, that there were bilateral diaphragmatic defects, and later, that there was probably enough esophageal length to permit repair.

CONCLUSION

1. An unusual case of bilateral diaphragmatic herniae in an infant is presented.
2. The advantages of the transthoracic approach are enumerated.
3. The need for early surgical repair is emphasized.
4. The importance of careful preoperative radiographic studies is stressed.

REFERENCES

1. Harrington, S. W.: Diaphragmatic Hernia of Children, Ann. Surg. **115:** 705, 1942.
2. Baumgarten, C. J., and Scott, R. F.: Surgical Emergency of Diaphragmatic Hernia in Infancy, Arch. Surg. 61: 170, 1950.
3. Lawler, R. H., West, J. W., and Lawler, E. G.: Treatment of Diaphragmatic Hernia in the Newborn, Am. J. Dis. Child. **84:** 79, July, 1952.
4. Sweet, R. H.: Esophageal Hiatus Hernia of the Diaphragm, Ann. Surg. **135:** 1-13, 1952.
5. Arnheim, E. E.: Congenital Hernia of the Diaphragm With Special Reference to Right Sided Hernia of the Liver and Intestines, Surg., Gynec. & Obst. **95:** 293, 1952.
6. Harrington, S. W.: Surgical Treatment of the More Common Types of Diaphragmatic Hernia; Esophageal Hiatus, Traumatic, Pleuroperitoneal Hiatus, Congenital Absence and Foramen of Morgagni, Report of 404 Cases, Ann. Surg. **122:** 546-568, **1945.**

LOCALIZED PULMONARY HYPERTROPHIC EMPHYSEMA

WILLIAM HENRY, M.D.
LYNWOOD, CALIF.

LOCALIZED pulmonary hypertrophic emphysema, unassociated with an aspirated foreign body or tenacious mucus from an acute respiratory infection, is an unusual condition which was barely mentioned in the medical literature prior to 1949. This disease usually occurs in infants and young children, although two cases discovered in the fourth decade of life have been reported. The exact etiology of the disease has not been established with certainty, but it appears reasonable to assume that it is congenital in origin. The diagnosis can usually be made radiologically if the existence of this entity is kept in mind. Surgical extirpation of the diseased pulmonary tissue appears to be strongly indicated. The following case report illustrates the diagnosis and management of pulmonary hypertrophic emphysema involving segments, or portions of segments, of the posterior segment of the right upper lobe, and the superior and lateral basal segments of the right lower lobe in a 10-year-old white boy.

CASE REPORT

T. G., a 10-year-old white boy, was first seen on Feb. 1, 1952, at which time a chest x-ray picture showed marked emphysema of the right lung.* When first seen the patient had recently recovered from a respiratory infection. A chest film had been ordered by his physician to determine if any pulmonary pathology was present. The patient gave an equivocal history of possible recent foreign body aspiration. The films (Figs. 1 and 2) were interpreted as showing obstructive emphysema caused by a nonopaque foreign body in the right bronchial tree. He was admitted to the St. Francis Hospital, Lynwood, where bronchoscopy was performed. This revealed an entirely normal tracheobronchial tree.

Following this procedure, a right Lipiodol bronchogram was performed on Feb. 6, 1952, and showed what was interpreted as a large air-filled, cystic area in the posterolateral lung field (Figs. 3 and 4).

A right thoracotomy was performed on Feb. 13, 1952, at which time three separate areas of pulmonary emphysema involving the posterior segment of the right upper lobe, the superior segment of the right lower lobe, and the lateral basal segment of the right lower lobe were found. The emphysematous areas compressed the normal pulmonary tissue. An artist's conception of the operative findings is shown in Fig. 5.

Inasmuch as there was some doubt as to whether entire segments were involved, it was thought feasible to attempt resection from the periphery inward. The line of demarcation with the normal lung was quite clear. A pleural incision was made around the periphery of the emphysematous area in the superior segment of the right lower lobe. Using gentle gauze pledget dissection, the intersegmental plane was easily developed. Intersegmental vessels were divided between mosquito clamps and ligated with fine silk, as were vessels at the hilar aspect of the emphysematous area. The bronchus to the emphysematous area was about 2 mm. in diameter; this was closed with fine silk. The other

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*The patient referred by Dr. Harold Mourer, Bellflower, Calif.

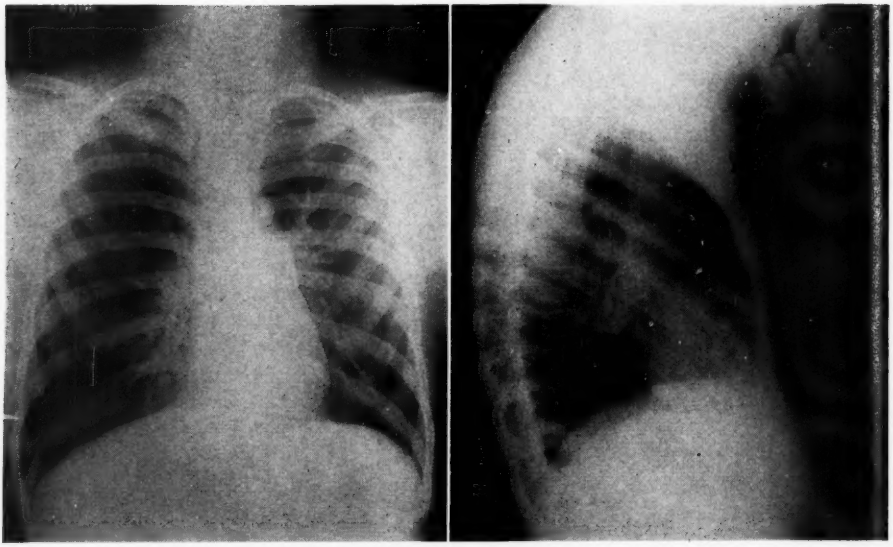

Fig. 1. Fig. 2.

Fig. 1.—Posteroanterior view of chest showing emphysema of the right lung.
Fig. 2.—Right lateral view of chest.

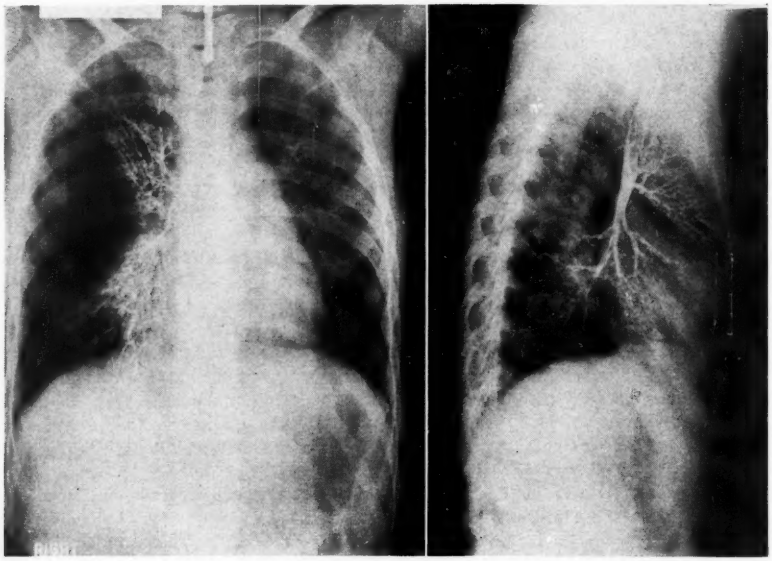

Fig. 3. Fig. 4.

Fig. 3.—Posteroanterior view of Lipiodol bronchogram showing area of emphysema.
Fig. 4.—Right lateral view of Lipiodol bronchogram showing area of emphysema.

two areas were then removed by a similar technique. Raw pleural surfaces were then closed with continuous fine catgut sutures to minimize air leaks. The middle lobe was anchored to prevent rotation and atelectasis. At the conclusion of the procedure there was satisfactory expansion of the lung with minimal air leaks. As the right lung did not entirely fill the pleural cavity, the phrenic nerve was crushed at the diaphragm to help de-

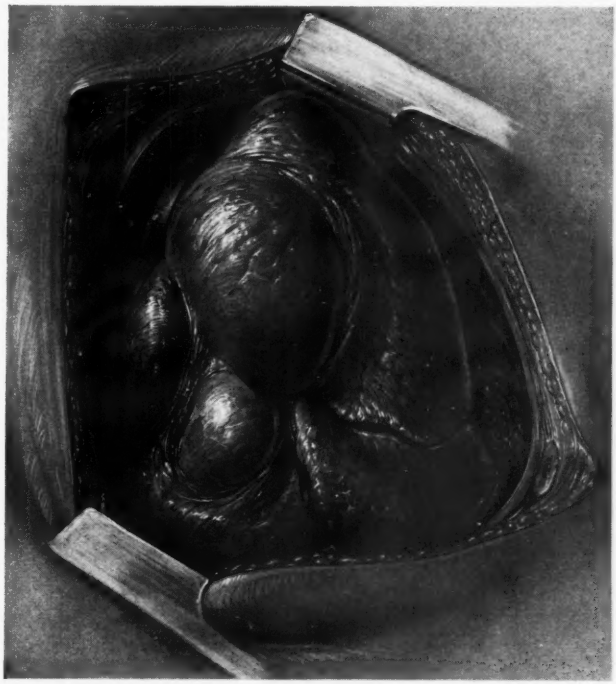

Fig. 5.—Artist's conception of operative findings.

Fig. 6.—Photograph of formalin-hardened specimens.

crease the size of the pleural cavity. Two intercostal catheters were connected to water-seal drainage. The patient had an uncomplicated postoperative course, and was discharged on the eighth postoperative day.

Photographs of the formalin-hardened segments are shown in Fig. 6. Microscopic sections of the surgically removed areas showed pulmonary emphysema, chronic bronchitis, and interstitial pneumonitis. Fig. 7 is a photomicrograph showing the emphysema.

The patient's progress has been followed for over a year and, he is entirely well. The right diaphragm is functioning. The vital capacity has increased from a preoperative reading of 1.7 L. to a recent postoperative reading of 3 L. A review of the patient's history revealed possible asthma prior to a tonsillectomy at the age of 3½, but none following this procedure. He was never allergic to any material except possibly barber's talc.

The most recent postoperative film of the chest is shown in Fig. 8.

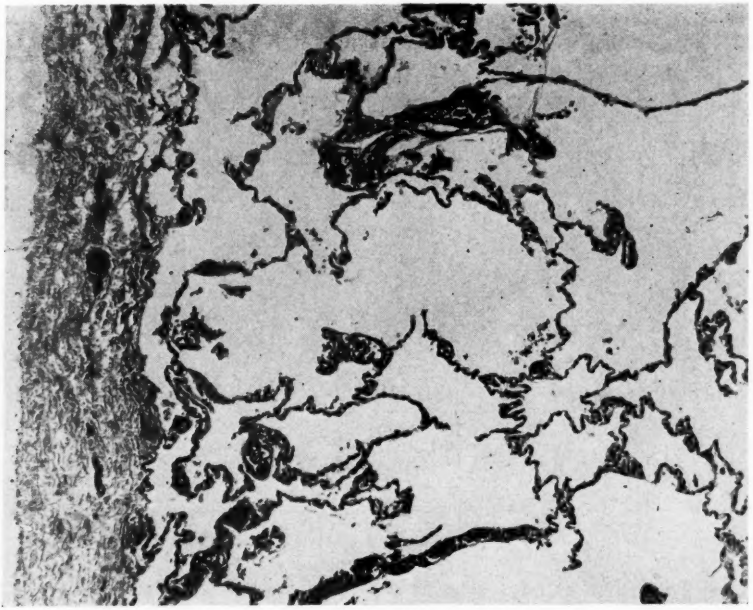

Fig. 7.—Photomicrograph showing emphysema typical of all specimens.

DISCUSSION

Historical.—The first recorded case was that of Royes[1] in 1938. He reported a large area of hypertrophic emphysema involving a portion of the right middle lobe, discovered during an autopsy on a 39-year-old man, who died following an accident. Royes found a flap of mucosa which partially occluded the bronchus to the affected area.

Leahy and Butsch,[2] in 1949, reported a case involving the left upper lobe in an 11-week-old white boy. This infant had marked respiratory distress from birth. A bronchoscopy was performed which was negative. Thoracotomy was performed with a preoperative diagnosis of probable cystic disease

of the lung. At operation a markedly distended left upper lobe and a collapsed lower lobe were noted. Left upper lobectomy was performed and the patient recovered.

Sweet,[3] in 1950, reported a case involving the left upper lobe in a 34-year-old man. The presenting complaint was dyspnea on exertion which had been present for twenty years. A left pneumonectomy was performed. In addition to the emphysema, sarcoidosis was present in the hilar glands as well as in the pulmonary tissue. This patient recovered.

Robertson and James,[4] in 1951, reported five cases of emphysema which they felt was congenital in origin, and in which the emphysema involved an entire lobe. They felt the emphysema was caused by folds of mucosa, or weakness of the bronchial wall forming a check-valve mechanism. The infants ranged in age from 2 weeks to 11 months; three cases had involvement of the right upper lobe, one the right middle lobe, and one the left upper lobe. In three cases in which lobectomy was performed all patients recovered, the other two patients not operated on died.

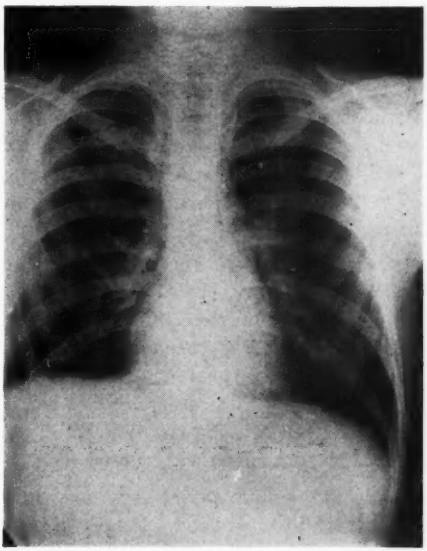

Fig. 8.—Posteroanterior view of chest taken four months postoperatively.

Shaw,[5] in 1952, reported two occurrences: one in a 4-year-old child involving the "sub-apical segment" of the left upper lobe, in which a segmental resection was performed with recovery; and one in a 7-week-old infant involving the right middle lobe, in which a middle lobectomy was followed by recovery of the patient.

Williams,[6] in 1952, reported a case involving the right middle lobe of a 17-month-old infant; a middle lobectomy was followed by the recovery of the patient.

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Diagnosis.—Most of the other recorded cases of localized pulmonary hypertrophic emphysema occurred in infants. The initial impression in this case, as in Williams's case,[6] was that the patient had obstructive emphysema caused by an intrabronchial foreign body, and bronchoscopy was carried out in an effort to remove the suspected foreign body. Lipiodol bronchogram following the bronchoscopy was of great assistance in determining the need for thoracotomy. If the possibility of localized pulmonary hypertrophic emphysema is borne in mind the correct preoperative diagnosis is possible.

Pathology.—Shaw[5] has given an excellent description of the pathology. He states that "the diseased segment is enlarged to many times its normal volume in a uniform dilatation of the air sacs. The bronchi, arteries, and veins are much smaller than one would expect to find supplying a segment of lung. Even when the bronchi are opened and the segment of lung tissue is removed from the body it does not collapse appreciably. It is uniformly pink in color even in adults, indicating that it has not taken part in the usual ventilation of lung tissue. Except for the pink color, it more closely resembles Gelfoam than any other substance. Several hours after being removed from the body, this tissue will float upon water with less than a tenth of its volume being submerged. Microscopic examination of the tissue reveals air sacs and alveoli that are generally distended and empty. The intervening septae are thin and there is no inflammatory reaction. The blood vessels are small and have thin walls. The bronchioles are empty. The bronchial mucosa is normal except for slight chronic inflammatory infiltration. The cause of the emphysema is not evident in the examination of the specimens."

In the present case, microscopic sections showed evidence of chronic bronchitis. During the two weeks that the patient was under observation prior to thoracotomy, he was afebrile, had a normal blood count, and no respiratory symptoms other than dyspnea on exertion. No further areas of emphysema have developed at the present time, more than one year after surgery. At operation the abnormal lung tissue was extremely soft and spongy. The color was pale pink, it did not deflate on releasing endotracheal pressure, and no emphysematous blebs were noted on the pleural aspect.

Etiology.—This condition should not be confused with regional obstructive emphysema in infants and children caused by thick viscid mucus as a result of acute respiratory infections.

Caffey,[7] in 1940, reported seven cases of this type of emphysema associated with acute respiratory infections in which cystic dilatations could be seen in roentgenograms, and which returned to normal with nonoperative treatment.

Obstructive emphysema of a lobe or a lung is commonly caused by an aspirated foreign body.

In the particular type of emphysema under discussion, most of the previous authors feel, as I do, that it is congenital in origin. Folds of mucosa producing valvelike mechanisms in the bronchus, compression of a thin-walled

bronchus by an adjacent pulmonary vessel, and localized bronchial chondromalacia have all been described as the underlying cause. No definite conclusions as to the etiology can be drawn from this case.

SUMMARY

Multiple resection from the periphery inward in a 10-year-old boy having pulmonary hypertrophic emphysema localized to segments or portions of segments of the posterior segment of the right upper lobe, superior segment of the right lower lobe, and lateral basal segment of the right lower lobe is reported. This is apparently the tenth recorded case of localized pulmonary hypertrophic emphysema occurring in infancy or childhood; two other examples were reported in adults. Surgical excision appears indicated in this condition.

REFERENCES

1. Royes, K.: Localized Pulmonary Hypertrophic Emphysema, Brit. M. J. 2: 659, 1938.
2. Leahy, L. J., and Butsch, W. L.: Surgical Management of Respiratory Emergencies During First Few Weeks of Life, Arch. Surg. 59: 466, 1949.
3. Sweet, R. H.: Case Records of Massachusetts General Hospital, New England J. Med. 242: 149, 1950.
4. Robertson, R., and James, E. S.: Congenital Lobar Emphysema, Pediatrics 8: 795, 1951.
5. Shaw, R. R.: Localized Hypertrophic Emphysema, Pediatrics 9: 220, 1952.
6. Williams, Mark H.: Localized Pulmonary Hypertrophic Emphysema, J. THORACIC SURG. 24: 522, 1952.
7. Caffey, J.: Regional Obstructive Emphysema in Infants and in Children, Am. J. Dis. Child. 60: 596, 1940.

VARIATIONS IN PULMONARY VASCULAR PRESSURES

STEVEN M. HORVATH, PH.D., AND D. M. MACCANON, M.S.*
IOWA CITY, IOWA

AN ABNORMAL type of pressure wave was observed during the course of an experiment wherein the "pulmonary capillary" pressure was being measured as suggested by Hellems and his associates.[1] This pressure wave grossly resembled an intrapleural pressure curve. Autopsy of this animal confirmed the presence of the catheter in the peripheral arterial vessel but immediately raised questions as to both the significance of this experimental method and the validity of the pressure values secured. Pressure measurements were then made in various vessels of the thorax accessible by the catheterization procedure being employed, in anticipation that the information obtained would at least partially resolve doubts about the method.

METHODS

Mongrel dogs were anesthetized with Nembutal, 30 mg. per kilogram intravenously. Catheters, No. 7 or No. 9, were passed under fluoroscopic visualization. Two catheters were placed on the left side, one in the aorta or left ventricle and the other in the left atrium or one of the pulmonary veins. Similarly on the right side one catheter was positioned in the pulmonary artery and another was wedged in a peripheral pulmonary arterial branch as described by Hellems and his associates.[1] In some instances a femoral artery was also cannulated. Patency of the catheters was assured by slow infusion or an occasional flushing with heparinized saline. Pressures were recorded simultaneously by means of Statham strain gauges and Sanborn or Brush oscillographs. Following a series of measurements at the above described positions, one or more catheters were moved so as to obtain records from additional places in the heart or adjacent vessels. This maneuver permitted the securing of various combinations of records from the right and left sides within a few heart beats. Usually the catheters were moved while the recordings of pressure were being made continuously and their positions then checked fluoroscopically. In several instances the catheter positions were confirmed at autopsy.

RESULTS

A large number of records was obtained from nine dogs on eleven occasions. A summarization of pertinent data obtained from these records is presented in Table I, and some tracings are reproduced in Figs. 1 and 2. The pressures obtained from the left atrium, pulmonary veins, and "pulmonary

From the Department of Physiology, College of Medicine, State University of Iowa.
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TABLE I. PRESSURE RELATIONSHIPS IN VESSELS OF THE THORACIC CAGE*

	FEMORAL ARTERY		AORTA		LEFT VENTRICLE		LEFT ATRIUM	PULMONARY VEIN		PULMONARY† CAPILLARY	PULMONARY ARTERY		RIGHT VENTRICLE		RIGHT ATRIUM	
	SYS.	DIAS.	SYS.	DIAS.	SYS.	DIAS.	MEAN	SYS.	DIAS.	MEAN	SYS.	DIAS.	SYS.	DIAS.	SYS.	DIAS.
							5.0			4.7	10	5				
							2.7			5.8						
			153	120	170	5	1.5	7.5	3.0	3.4	15	8	18	−1	−1.5	−2.5
	180	120	138	115	150	1	3.0	10.5	5.0	3.3	13	6	13	1	0.5	−0.5
	152	108	135	125	156	7	3.0	13.0	1.0	2.0	32	8	31	−1	1.0	−1.0
			128	105	165	11	4.0	4.0	3.2	3.0	10	4	12	1	1.0	−1.0
							9.0	12.0	9.0	9.0	15	7	15	0		
			140	119	160	5	7.5	14.0	11.0	8.0	14	8	15	−1	2.5	−1.5
			136	115	140	5	6.0	8.0	0.9	6.0	15	7	18	2	2.0	−2.0
			134	110	144	4	5.0	12.0	8.0	8.0	20	10	20	0	7.0	3.0
	105	70					5.0	10.0	3.0	7.0	12	4	12	1		
Mean			138	116	156	6	4.7	10.1	4.9	5.5	15.6	6.7	17.1	0.2	2.1	−0.8

*All pressures are expressed in mm. Hg, eleven experiments.
†Wedged pulmonary artery.

capillary" are of greatest interest. The mean left atrial pressure exceeded the mean "pulmonary capillary" pressure by some 0.8 mm. Hg, but this relation was not consistently evident in individual cases. In three instances, a higher value was observed in the left atrium than the pulmonary capillary by 0.3 to

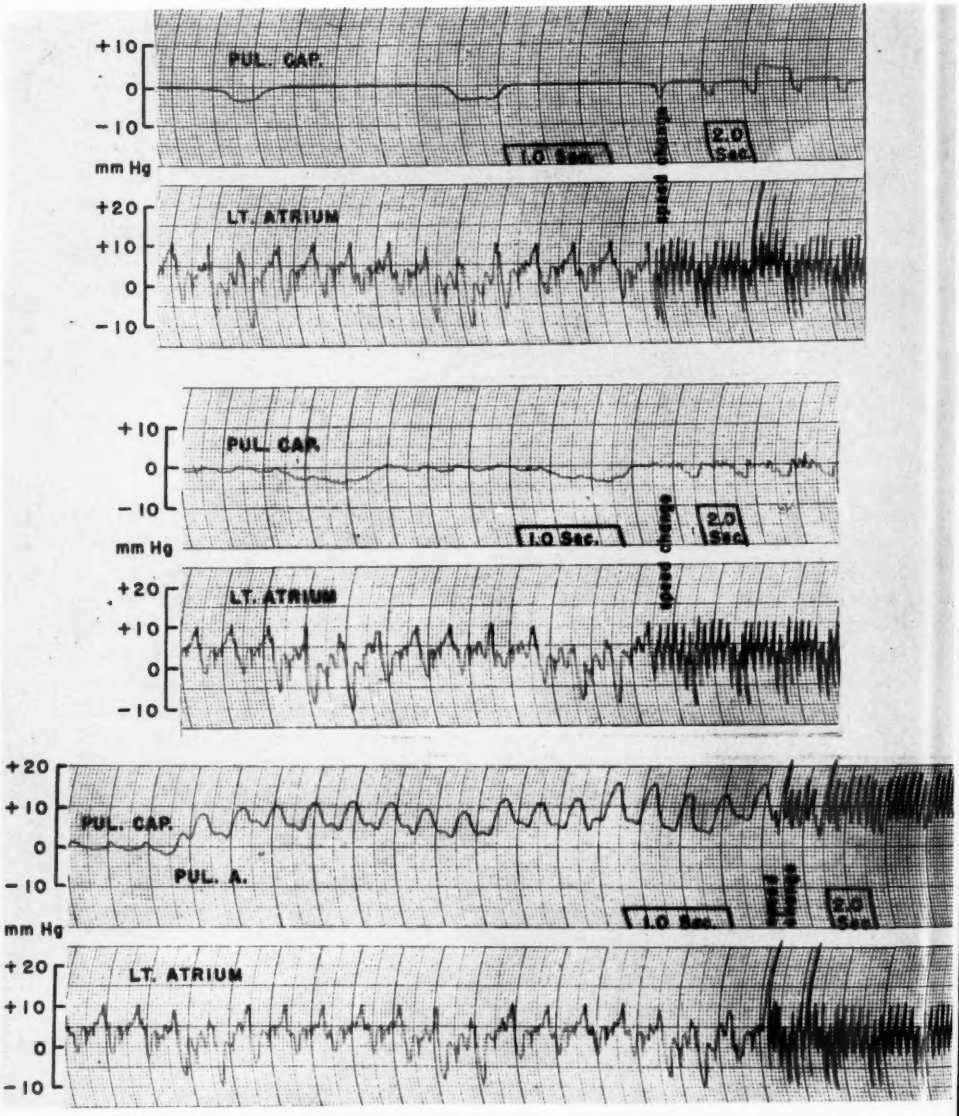

Fig. 1.—A type of pulmonary "capillary" pressure sometimes observed is illustrated. The lowest record shows the transition to pulmonary arterial pressures as the catheter was being withdrawn. The left atrial pressure is presented for reference.

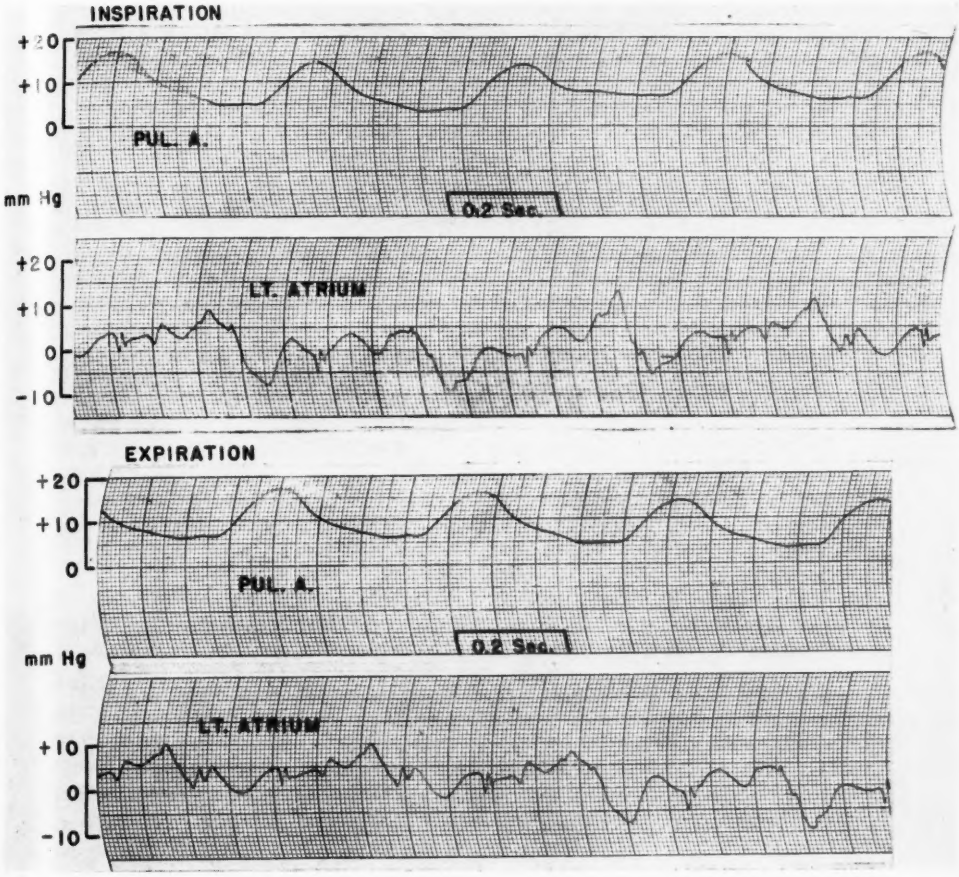

Fig. 2.—Fast record, demonstrating pattern of left atrial pressure tracings with pulmonary arterial waves as reference.

1.0 mm. Hg. On two occasions the pressures were equivalent, while in the remainder the pulmonary capillary values exceeded those observed in the left atrium by 3 to 0.3 mm. Hg.

Although these differences are small and the technical difficulties of recording great, some doubt is held concerning the validity of using "wedged catheter pressures" as representative of left atrial pressure. On three occasions a record similar to that shown in Fig. 1 has been obtained. In each instance the catheter was positioned in a vessel in the lower border of the right lung lobe. Can the recorded pressure be considered a "pulmonary capillary" pressure? It appears to be a record of intrapleural pressure; withdrawal of blood was not possible when the catheter was in this position. Slow withdrawal of the catheter from this position eventually resulted in a pressure

tracing somewhat similar to that of a "pulmonary capillary" pressure. However, in Fig. 1 the transition to a modified pulmonary arterial pressure is too rapid to permit positive identification. When a catheter is wedged as far out in the periphery of the lung as these were, the rapid injection of 1 ml. of saline induced a marked elevation of pressure which persisted for several minutes before it gradually returned to its former level. Autopsy observations failed to show any injury to vessels or lung tissue as a consequence of this procedure.

The pressure tracings (Fig. 2) obtained from the left atrium through a catheter going down the carotid artery and lying within the left atrium are somewhat similar in form to those published by Ankeney,[2] but the instantaneous pressure values are somewhat different. There are a number of negative values on the curve, especially noticeable during inspiration. Although some artifacts may be present due to catheter vibrations of intracardiac origin, they are still suggestive of pressure alterations occurring in the intact closed-chest animal.

Pressures measured when a catheter is apparently wedged into a pulmonary vein were invariably greater than simultaneous measurements of pulmonary capillary and are similar to the pulmonary arterial pressures. However, artifacts prevented accurate determinations of points of similarity in the two curves.

DISCUSSION

The occasional observation of an intrapleural type of pressure wave when a catheter is wedged into a distal pulmonary artery was an unexpected finding. Animals in which the phenomenon was observed were narrow chested and the catheter was placed far out into the diaphragmatic border of the lung field. The small mass of tissue present at this point may have permitted the transmission of intrapleural pressure variations but it is inconceivable how such an event could occur in view of the pressure gradient theoretically existing. Regardless of the causative factors leading to such pressure tracings, their occurrence does suggest that "pulmonary capillary pressures" must be evaluated with considerable caution.

Pulmonary capillary pressures slightly higher than left atrial pressures, as observed in this study, are in general agreement with the similar, although greater, differences reported by Ankeney[2] in the open-chest dog. Gorlin and his co-workers,[3] however, were unable to find any significant differences in pressure between these two sites and suggest that within the error of measurement the values are equal. Weissel and his associates,[4] Lagerlöf and Werkö,[5] and Cournand[6] have, on the basis of studies made in cases with atrial septal defects, expressed doubt as to the pressures recorded by the method of wedging being "capillary pressure" and implied that it may represent a modified pulmonary venous pressure.

However, Calazel and his fellow workers[7] were unable to obtain pressure curves suggestive of this venous character by obstructing a peripheral pulmonary artery in five of their six patients. The pulmonary capillary pressure curves obtained in the present study cannot be used to confirm or deny these

observations, since in the majority of instances they were essentially flat waves with minor and inconstant phasic variations. This does not, however, preclude the possibility that a manometric system consisting of pulmonary capillaries and venules is formed when the catheter is wedged in a suitable peripheral pulmonary arteriole. The reverse manometer system (pulmonary capillary arteriole), formed when a catheter is wedged into a pulmonary vein postulated by Lagerlöf and Werkö,[5] of capillaries and arterioles appeared to be present in the study being reported. The pulmonary venous pressures (PCA) were higher than those observed in the left atrium but lower than those of the pulmonary artery. These findings agreed also with those reported by Weissel and his associates,[4] but are in contrast to the results of Hellems and his associates,[8] who found identical mean values on both sides of the capillary bed.

SUMMARY AND CONCLUSIONS

The determination of "pulmonary capillary pressure" is not a simple matter of obtaining a pressure recording while a catheter is wedged into a peripheral pulmonary artery. If the catheter is wedged into a suitable arterial vessel, respiratory pressure variations similar to intrapleural pressures may be recorded. The possibility that under certain circumstances "pulmonary capillary" pressures really represent pulmonary capillary venule pressures cannot be ignored, since pressures recorded from a wedged pulmonary vein were of such magnitude as to indicate the possibility of the establishment of a pulmonary capillary arteriole manometric system.

REFERENCES

1. Hellems, H. K., Haynes, F. W., Dexter, L., and Kinney, T. D.: Pulmonary Capillary Pressure in Animals Estimated by Venous and Arterial Catheterization, Am. J. Physiol. **155**: 98-105, 1948.
2. Ankeney, J. L.: Interrelations of Pulmonary Arterial, 'Capillary' and Left Atrial Pressures Under Experimental Conditions, Am. J. Physiol. **169**: 40-49, 1952.
3. Gorlin, R., Dow, J. W., Hellems, H. K., and Dexter, L.: Pulmonary "Capillary" Pressure as an Index of Left Atrial Mean Pressure in Dogs, Am. J. Med. **8**: 396, 1950.
4. Weissel, W., Salzmann, F., and Vetter, H.: Pulmonary Capillary Arterial Pressure Pulse in Man, Brit. Heart J. **14**: 47-52, 1952.
5. Lagerlöf, H. and Werkö, L.: Studies on Circulation of Blood in Man; the Pulmonary Capillary Venous Pressure Pulse in Man, Scandinav. J. Clin. & Lab. Invest. **1**: 147-161, 1949.
6. Cournand, A.: Some Aspects of the Pulmonary Circulation in Normal Man and in Chronic Cardiopulmonary Diseases, Circulation **2**: 641-657, 1950.
7. Calazel, P., Gerard, R., Daley, R., Drapner, R., Foster, J., and Bing, R. J.: A Comparison of the Right and Left Auricular, Capillary and Pulmonary Artery Pressures in Nine Patients with Auricular Septal Defect, Bull. Johns Hopkins Hosp. **88**: 20-37, 1951.
8. Hellems, H. K., Haynes, F. W., and Dexter, L.: Pulmonary 'Capillary' Pressure in Man, J. Appl. Physiol. **2**: 24-29, 1949.

BILATERAL GRANULOMA OF THE LARYNX DUE TO INTRATRACHEAL ANESTHESIA

DERMONT W. MELICK, M.D., AUDREY G. URRY, M.D., AND JOHN B. MCADAMS, M.D.
PHOENIX, ARIZ.

THE administration of anesthesia by way of an endotracheal tube has repeatedly been recognized as one of the major advances in anesthesia and thoracic surgery. It allows the anesthetist to maintain the patient in a state of "physiologic safety" by reason of this endotracheal anesthetic technique.

It has always been amazing, however, that patients do not have more laryngeal difficulty. The laryngeal tissues have always been recognized as delicate and extremely sensitive. It would be suspected that the passage of a rubber tube through these delicate sensitive tissues followed by the rather prolonged contact with this foreign body would result in a certain amount of irritation. The most skillful intubation could not logically be expected to remove all the factors that have to do with laryngeal irritation. The incidence of mild traumatic postoperative laryngitis is not known. Individuals exhibiting voice changes or "sore throat" following endotracheal anesthesia are known to all of us, but the frequency is not a matter of record. Tracheal web formation has been reported in the *New England Journal of Medicine* as a result of intubation.[2]

Trauma to the vocal cords of enough intensity to cause formation of granulomatous lesions is a rare occurrence. In the present literature there are twenty-three cases reported, of which thirteen were bilateral granuloma and ten were unilateral granuloma. Curiously enough, in one series of ten cases, nine were reported to be in women.[1] The case reported here also occurred in a woman. This sex predominance may mean that women have smaller voice boxes (could overuse be a cause of hypoplasia?) or the tissues are more delicate.

CASE REPORT

Mrs. D. S., a 28-year-old woman, was a patient at the Arizona State Sanatorium. A right upper lobectomy for tuberculosis was performed on Dec. 9, 1950. The anesthesia time (from intubation to extubation) was four hours and twenty minutes. After surgery she complained of a sore throat and her voice was hoarse. The hoarseness persisted and eight weeks following lobectomy, direct laryngoscopy revealed polypoid tumor masses on either vocal cord. The granuloma of the right vocal cord was somewhat larger, so it was removed first. Fourteen days later the polyp was removed from the left vocal cord.

The pathologic examination and report by Dr. Harold Wood was identical in each instance: "The gross specimens consist of small bits of gray-brown tissue which vary from 0.2 to 0.6 cm. in diameter. The microscopical examination reveals a very active polypoid growth of vascular granulation tissue, which is superficially ulcerated and inflamed. It contains mitoses and simulates a capillary hemangioma or granuloma pyogenicum. There is no evidence of malignancy. Diagnosis: A polypoid capillary hemangioma or a very vascular laryngeal polyp."

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With reference to the intubation and anesthesia in this patient, the co-author (A. G. U.) comments as follows: "The intubation was not traumatic and exposure of the larynx and insertion of the tube was done without difficulty. Pentothal Sodium, nitrous oxide, and curare were used. After the initial depth of anesthesia for intubation, the patient was maintained in plane one. During the last hour she was maintained so lightly in plane one that she swallowed and coughed occasionally. She was awake, talked, and was oriented in the operating room immediately after the tracheobronchial toilet. Undoubtedly the too light anesthesia during the last hour with the motion of the larynx over the tube due to swallowing and coughing is the trauma responsible for the production of the granulomatous tissue."

The patient was discharged from the sanatorium three and one-half months following lobectomy and one month after removal of the second granuloma. On discharge from the sanatorium the vocal cords had a normal appearance and her voice had returned to normal. A recheck by an otolaryngologist in another city four months following discharge from the sanatorium revealed the vocal cords to be entirely normal.

COMMENT

Lieberman[1] of Philadelphia has this to say with relation to this condition. "Granuloma of the larynx following intratracheal anesthesia is probably occurring more frequently than the literature would indicate, and perhaps more care should be taken. A careful history is important. Particular attention should be paid to a history of recurrent laryngitis, and if this is elicited, possibly intratracheal anesthesia should not be used. The tube certainly should be passed under direct vision and with a minimum of trauma and only when the vocal cords are wide open. The tube should be sterilized just before using. The tube should be passed by a well-trained anesthetist, and great care should be taken to avoid undue movement of the tube during the operation."

SUMMARY

A case of bilateral granuloma of the larynx is reported here. The causation of this bilateral granuloma has been assumed to be due to the movement of the larynx over the endotracheal tube during too light a plane of anesthesia.

REFERENCES

1. Lieberman, G. E.: Bilateral Granuloma of the Larynx Due to Intratracheal Anesthesia, Arch. Otolaryng. **56**: 2-4-206, 1952.
2. Etsten, Benjamin, and Mahler, Donald: Subglottic Membrane, New England J. Med. **245**: 957-960, 1951.

Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 34th annual meeting of the American Association for Thoracic Surgery will be held May 3, 4, and 5, 1954, in Montreal, Que., Canada. Headquarters will be at the Sheraton-Mount Royal Hotel.

Requests for Hotel Reservations

These should be mailed *directly* to the Reservation Manager, Sheraton-Mount Royal Hotel, Montreal, Que., Canada. Please mention this Association, type of accommodation desired, date, and approximate hour of arrival. If accommodations are desired elsewhere, please communicate directly with hotel of your choice.

Thoracic Surgical Forum

After due consideration, the Council has decided to try an experiment by assigning one morning session to a "Thoracic Surgical Forum" for the presentation of short papers on current thoracic research, anatomy, modified surgical techniques, etc. Papers at this session will be limited sharply to ten minutes (including the showing of lantern slides or other illustrative material) with three minutes as the maximum discussion of each paper.

These papers will be published together in one issue of THE JOURNAL OF THORACIC SURGERY. Maximum length: 3,000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

Abstracts for Papers

Abstracts for the presentation of papers at the 1954 meeting, including papers before the Forum, must be received on or before January 1, 1954, otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Forum" or "For Regular Program."

Five, repeat five, copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words and accurately reflect the contents of the completed paper.

Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee not later than Jan. 1, 1954, to be considered for action in Montreal. Applications received beyond this deadline will be deferred for consideration until 1955.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the sponsoring statement on the application form, the sponsors *must* submit a separate letter concerning the applicant direct to Dr. Lyman H. Brewer, III, Chairman of the Membership Committee, 2010 Wilshire Blvd., Los Angeles, Calif. Completed application forms should likewise be sent to Dr. Brewer. These regulations are at the direction of the Council and were made effective in 1952 at Dallas.